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Growth Through Agricultural Progress

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Paul R. Miller

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DISTRIBUTION OF THE RENIFORM NEMATODE IN RELATION TO CROP FAILURE OF COTTON IN LOUISIANA[1]

W. Birchfield[2] and J. E. Jones[3]

Summary

Reniform nematode, Rotylenchulus reniformis Linford & Oliveira, 1940, was previously known in only a few Louisiana cottonfields. In a recent survey it was found associated with cotton failures distributed over 2000-2500 acres in two Parishes. Reniform nematode has not been considered as a virulent pathogen of any particular crop plant, although it has a wide host range. In Louisiana it was constantly associated with severe disease symptoms. The symptoms were dwarfing, premature decay with loss of secondary roots and death of young cotton. These conditions resulted in poor stands, grassy areas and ultimate yield reduction of 40 to 60% of the crop.

The reniform nematode, Rotylenchulus reniformis Linford & Oliveira, was first described from roots of cowpea from Hawaii in 1940 (3). Simultaneously, in a separate publication by Linford and Yap (4), its host range was reported to embrace 65 plant species in 30 different families. Also in 1940, this nematode was identified as a parasite of cotton in Georgia by G. Steiner as reported by Smith (10). In 1941 Smith and Taylor (11) reported that it had been found on roots of both cotton and cowpeas from Baton Rouge. It was next reported (7) from cotton in Louisiana along the "Perkins Road Farm," also the property of the University and located a few miles south of the old agronomy farm. Subsequently it was found on cabbage and beets in Rapides Parish, Louisiana. Martin (5) recently reported it on sweetpotato in Louisiana. Neal suggested (7) that reniform nematode is "an important factor in the incidence of Fusarium wilt" of cotton in Louisiana. Jones, et al. (2) and Newsom and Jones (8) showed in greenhouse and field experiments with different cotton varieties and fumigation studies in soil infested with reniform nematode that this parasite caused an appreciable yield reduction of varieties tested. In addition to reducing cotton yield by 30 to 50%, this nematode also caused a delay in maturity, a reduction in size of boll, and in some years a reduction in lint percentage. They obtained satisfactory control of this nematode by soil fumigation using dichloropropane-dichloropropene at 9 and 13 gallons per acre, ethylene dibromide (83%) at 4 gallons per acre and 1,2-dibromo-3-chloropropane at 1/2 gallon per acre.

Peacock (9) reported light infestation of the reniform nematode on Gossypium spp. on the Gold Coast of Africa, and Minton and Hopper (6) reported its occurrence in a cotton field in Alabama.

During the last few years certain cotton farmers in Rapides and Avoyelles Parishes experienced 40 to 60% reduction in yields. These losses were not generally associated without regard to the variety employed. Although the cotton disease complex in that area usually involved root-knot and Fusarium and Verticillium wilts (1), these diseases were not implicated in all cases. A limited survey of some of the problem fields was begun during the fall of 1960. Approximately 100 samples were analyzed. Thirty percent of these were found to be infested by the reniform nematode. In many samples large populations of reniform nematode were found whereas other plant-parasitic forms were present in small numbers. In some samples the reniform nematode occurred with root-knot nematode larvae but in all such instances the reniform nematode predominated.

Disease symptoms associated with the reniform nematode on cotton in the field were abnormally short plants and dead or unhealthy plants with prematurely decayed root systems. This resulted in grassy areas in the field where the organism was present. Typical field symptoms and egg masses produced by the reniform nematode on cotton roots are shown in Figure 1. Margins of infection in cotton fields were easy to observe. These boundaries were marked by the farmers for soil fumigation. Reniform nematode populations were usually large even after spring plowing when populations of plant-parasitic nematodes are usually low. Suggested treat-

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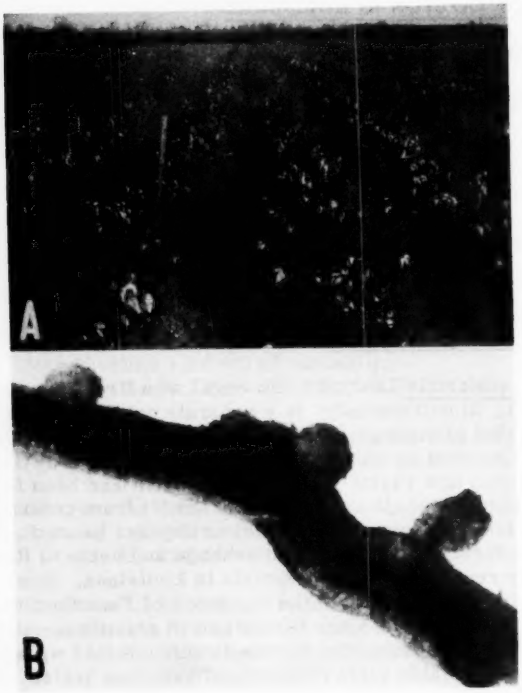

FIGURE 1. A -- A typical infested field showing stunted cotton in a grassy field. Yield losses were severe in such fields. B -- Egg masses of the reniform nematode on cotton roots. (Photograph by J. H. Roberts)

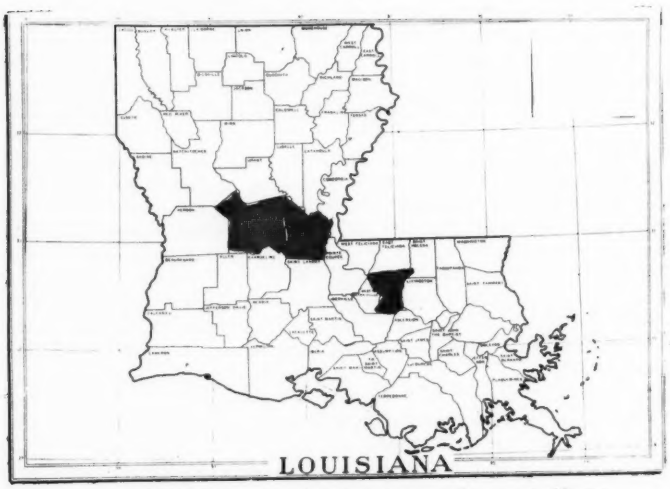

FIGURE 2. Parish locations where reniform nematode was found in Louisiana.

ment for these problem spots was 1/2 gallon of 1,2-dibromo-3-chloropropane per acre applied in the rows the following year.

In Louisiana the reniform nematode was found in East Baton Rouge, Rapides and Avoyelles Parishes (Fig 2). Parts of St. Landry Parish were included in the survey but all samples were negative. Heaviest infestations were found near Bunkie and Cheneyville on both sides of Bayou Boeuf in Avoyelles and Rapides Parishes. A serious infestation was also observed in several fields involving considerable acreage in the Boyce area in Rapides Parish.

Approximate cotton area infested by this pest was estimated by Ben Simpson, the county agent of Rapides Parish, to be about 2000 to 2500 acres where yield reductions were serious. This area, which averages about 2 bales per acre, is the best producing land in the State. Previously, reniform nematode was considered only a mild parasite not associated with any particular crop failure in Louisiana. However, in view of the results of the present survey, the areas involved and its constant association with disease and losses, reniform nematode must be considered a severe pathogen.

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CONTROL OF PEANUT LEAF SPOT (CERCOSPORA ARACHIDICOLA HORI) BY FUNGICIDAL SPRAYS

W. E. Cooper

Summary

Five fungicides, TC-90, maneb, dodine, Phaltan and Dyrene, which were applied as high volume sprays at weekly and biweekly intervals, were compared at two locations with the currently recommended biweekly application of sulfur - 4% (metallic) copper dust for peanut leaf spot control. The experimental liquid copper, TC-90, was effective at weekly and biweekly intervals and maneb and dodine were effective as shown by defoliation and yield responses when applied weekly.

INTRODUCTION

The effectiveness of sulfur and of sulfur-Tribasic Copper Sulfate (equivalent to 4% metallic copper) dusts in controlling the peanut leaf spot diseases (Cercospora arachidicola Hori and C. personata (Berk. & Curt.) Ell. & Ev.) and decreasing the losses caused by them was conclusively demonstrated in North Carolina (1) and Virginia (2) during the 1940's. Use of these fungicides was generally accepted by the growers in both States, however, the sulfur dust frequently causes intense eye irritation of the duster operator and this has limited its use.

Several organic fungicides which were available in 1951-1953 were much less effective than the sulfur-copper dust in controlling these diseases when applied as dusts at biweekly intervals, and not quite so effective when applied weekly (1). Grower acceptance of a weekly fungicide schedule was not considered likely. There is now a trend to sprayer application of pesticides to crops in the peanut-producing area. Since sprayer equipment is available to many growers, the objectives of the present study are an evaluation of: 1) the new fungicides for leaf spot control; and 2) the possibility of utilizing sprays for their application.

MATERIALS AND METHODS

The five experimental fungicides included in these 1960 tests, their active ingredient and the amount of the formulation in 100 gallons spray are as follows: 80% maneb (Dithane M-22) at two rates: 1.0 and 1.5 pounds; an experimental copper in an oil solution (TC-90) at 1.5 gallons; 50% N-trichloromethylthiophthalimide (Phaltan) at 2.0 pounds; 65% dodine (Cyprex) at 0.5 pound; and 50% 2,4-dichloro-6-(o-chloroanilino)-s-triazine (Dyrene) at 2.0 pounds. Rates (pounds/100 gallons) refer to the commercial formulations. Each fungicide was compared at weekly and biweekly applications for a total of six and three applications, respectively. The amount of the spray mixture applied increased from 100 to 150 gallons/acre as the plants increased in size; however, the concentrations remained constant. In addition to a non-treated check the currently recommended leaf spot control of sulfur - 4% copper dust (3) was included.

The tests were conducted at research stations at Rocky Mount and Lewiston, North Carolina. Fields that had been planted with the variety NC 2 were infested by scattering shredded, leaf spot infested, peanut hay at approximately 1000 pounds/acre about 2 weeks after the peanuts had emerged. Six replicates of 14 treatments in randomized blocks were used at each location. Each plot was a single row with a non-treated row between plots. Plot size was 30 ft. x 3 ft. at Rocky Mount and 45 ft. x 3 ft. at Lewiston.

A gasoline-engine powered, 30-gallon sprayer was equipped with a high pressure hose and a hand boom with three hollow-cone disc nozzles spaced 18 inches apart.

During the first application the wettable powder sprays did not satisfactorily wet the peanut leaves. This was largely corrected for the third and subsequent applications by adding 1.2 ml of Triton B 1956 per gallon of the wettable powder sprays.

As a measure of possible fungicide phytotoxicity the height of the mainstem of five plants, taken at given intervals within the row, was determined. To compare the effectiveness of the treatments in reducing defoliation, the leaves actually defoliated from the same five mainstems used for height measurements were counted. To evaluate the effect of the treatments on yield the plants were harvested, cured under field conditions and picked with conventional equipment. As an evaluation of quality a 2-quart sample of pods was drawn from each plot yield, weighed, mechanically shelled and the kernels passing over a 15/64-inch screen were weighed. On the basis of these weights, a shelling percentage was calculated.

Table 1. Effectiveness of peanut leaf spot control with weekly and biweekly fungicidal sprays as indicated by defoliation.

Fungicide	Rate/acre[a]	Number of leaves defoliated/mainstem			
		Rocky Mount 10/3/60		Lewiston 9/22/60	
		2-week	1-week	2-week	1-week
Nontreated	--	22.8	--	20.0	--
Sulfur-copper dust[b]	15-20 lb.	15.1	--	13.2	--
Maneb	1.0-1.5 lb.	21.6	15.7	17.0	12.1
Maneb	1.5-2.25 lb.	22.7	15.2	16.7	12.5
TC-90	1.5-2.25 gal.	16.3	8.9	11.4	9.0
Phaltan	2.0-3.0 lb.	21.9	21.2	19.2	14.3
Dodine	0.5-0.75 lb.	21.8	18.7	13.6	11.0
Dyrene	2.0-3.0 lb.	21.6	20.6	17.8	12.5
LSD between fungicides or intervals	.05	2.76		1.85	
	.01	3.67		2.47	
Coefficient of variation		13%		11%	

[a]Rates refer to the commercial formulations.
[b]The recommended peanut leaf spot control practice is included as a treated check.

Table 2. Effectiveness of peanut leaf spot control with weekly and biweekly fungicidal sprays as indicated by pod yields.

Fungicide	Rate/acre[a]	Pod yields (pounds/acre)			
		Rocky Mount 10/18/60		Lewiston 10/11/60	
		2-week	1-week	2-week	1-week
Nontreated	--	1936	--	968	--
Sulfur-copper dust[b]	15-20 lb.	3001	--	2023	--
Maneb	1.0-1.5 lb.	2357	2420	1517	2104
Maneb	1.5-2.25 lb.	2144	2773	1462	1984
TC-90	1.5-2.25 gal.	2735	2793	2049	2510
Phaltan	2.0-4.0 lb.	1946	2217	1107	1733
Dodine	0.5-0.75 lb.	2163	2545	1865	2233
Dyrene	2.0-3.0 lb.	2289	2628	1571	1959
LSD between fungicides or intervals	.05	335.3		266.1	
	.01	446.0		353.9	
Coefficient of variation		12%		13%	

[a]Rates refer to the commercial formulations.
[b]The recommended peanut leaf spot control practice is included as a treated check.

RESULTS

Early leaf spot caused by C. arachidicola was severe when the fungicide treatments were initiated with defoliation already evident at Lewiston. At no time was there more than a very light trace of late leaf spot caused by C. personata at either location. Observations following the treatments indicated only one instance of phytotoxicity. Within 2 hours after the third application at Lewiston a few young, but fully expanded, leaflets on all TC-90 treated plots became water-soaked and greenish brown. The affected leaflets later developed large yellowish white areas with some marginal burning. The temperature and humidity were very high at time of this application. There were no significant differences in the height of plants treated with the various fungicides at either location.

The mean defoliation values (Table 1) show that at Rocky Mount biweekly applications of only sulfur-copper dust and TC-90 significantly decreased defoliation; also weekly applications of maneb (both rates) TC-90 and dodine were effective. Defoliation was significantly less for the weekly than biweekly applications for maneb, TC-90 and dodine. At Lewiston all fungicide treatments except biweekly applications of dodine and Dyrene significantly reduced defoliation. Also, defoliation was less with weekly than biweekly applications for all fungicides.

Perhaps a more valid comparison would be with the effectiveness of sulfur-copper dust. At Rocky Mount TC-90 applied biweekly and maneb applied weekly were as effective as, and TC-90 at weekly intervals had less defoliation than, the sulfur-copper dust treatment. At Lewiston TC-90 and dodine applied biweekly and maneb, Phaltan, and Dyrene at weekly intervals were as effective as, and TC-90 and dodine at weekly intervals had less defoliation than the sulfur-copper dust. All other treatments at each location were more severely defoliated than the sulfur-copper treatment.

The average treatment yields (Table 2) show that sulfur-copper dust and TC-90 at biweekly and maneb, TC-90, dodine and Dyrene at weekly applications resulted in yields greater than non-treated at Rocky Mount. At Lewiston all biweekly applications, except Phaltan, and all weekly applications yielded more than the non-treated. Furthermore, all weekly applications gave yields greater than biweekly applications of the same fungicide.

Comparing yields with those of the copper-sulfur treatment, TC-90 at biweekly and TC-90 and the higher rate of maneb at weekly intervals at Rocky Mount and TC-90 and dodine at biweekly and maneb, dodine, and Dyrene applied weekly at Lewiston were as effective. Only weekly applications of TC-90 yielded more than the copper-sulfur treatment at Lewiston.

The mean shelling percentages of the pod samples from the various treatments showed no differences between treatments.

DISCUSSION

The results are based on the control of early leaf spot (C. arachidicola). The variety NC 2, used in these tests, is susceptible to this pathogen but relatively resistant to late leaf spot (C. personata). NC 2 is currently grown very extensively in North Carolina. Varieties differ in relative susceptibility to the two leaf spot pathogens, however, no fungicidal specificity has been reported for these fungi.

Only TC-90 was effective in a biweekly schedule, such as the growers have been using with sulfur-copper dust. Dodine showed some promise in this respect at Lewiston. Perhaps this material might be effective in a biweekly schedule under lower levels of infestation.

Regarding possible spray schedules with closer intervals than recommended for sulfur-copper dust, weekly applications of maneb were essentially equal to the recommended sulfur-copper schedule in controlling defoliation and increasing yields at both locations. There was no difference between the two rates. Therefore, applications at an interval between 1 and 2 weeks may be adequate with less severe infestation.

Of the other three materials, dodine, which was used at only 0.5 to 0.75 pound/acre, was promising at Lewiston. Further study of rates, concentrations, and intervals may be rewarding; however, its phytotoxicity may be a limiting factor with more concentrated sprays. Although Phaltan and Dyrene at weekly intervals reduced defoliation at Lewiston, their relative effectiveness is not encouraging.

Although none of the experimental fungicides indicated appreciable phytotoxicity at the spray concentrations used, further testing will be needed at concentrations necessary to apply the same quantity of fungicides at lower gallonage.

The question of residues of these experimental fungicides on foliage and pods remains. Maneb has been cleared for pods and kernels but not for the foliage to be used for hay. The liquid copper, TC-90, should not present special residue problems; however, the other three materials would probably require clearance even before use in extensive test demonstrations.

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A VIRUS LATENT IN SOME CUCURBITS

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Summary

A virus latent in PMR 45 cantaloup, Charleston Gray watermelon, Marketer cucumber, and Summer Straightneck squash was isolated from cantaloup selections growing at La Jolla, California. The virus induces small circular red-bordered necrotic lesions with light brown centers on Chenopodium amaranticolor 6 to 8 days after inoculation but does not become systemic. It is readily transmissible to susceptible hosts by mechanical means and by the aphid, Myzus persicae. Preliminary tests indicate that the virus is not persistent in the aphid. Physical properties can be described as follows: Infectious after 10 minutes at 50°C but not at 55°, at dilution of 1:100 but not of 1:1000, after 3 days' storage in vitro at 22° but not after 4. Preliminary host-range studies indicate that the virus is also infectious to the garden pea var. Laxton Progress, bean variety Pinto, and spinach variety Dixie Market but not infectious to Nicotiana glutinosa, N. tabacum (Samsun), N. clevelandii, Gomphrena globosa, Datura stramonium, California Wonder pepper, lettuce, sugar beet, garden beet and southern field pea var. Black. Studies indicate no apparent relationship between the latent virus and cucumber mosaic virus, squash mosaic virus, watermelon mosaic virus, beet mosaic virus, or lettuce mosaic virus.

During routine assays of viruses infectious to certain cucurbits, an unidentified virus was found in cantaloup plants growing at La Jolla, California. Specimens collected were suspected of being infected with the watermelon mosaic virus (WMV), the squash mosaic virus (SMV), or both. The virus was isolated by inoculation to a series of diagnostic hosts which included Chenopodium amaranticolor. This host, apparently immune from most isolates of WMV and SMV, developed small circular red-bordered necrotic spots with light brown centers 6 to 8 days after inoculation (Fig. 1). This report is concerned with preliminary studies relating to transmission, physical properties, and preliminary host range of the virus.

FIGURE 1. C. amaranticolor leaf 8 days after inoculation with the latent virus.

MATERIALS AND METHODS

Twelve specimens of cantaloup selections were collected from the breeding plot at La Jolla, California and airmailed to Beltsville, Maryland. Tissue from each specimen was ground with a mortar and pestle in a 0.5% buffer solution (pH 7.8) and the inocula applied with sterilized cheese cloth pads. Inoculations by mechanical means were made to cotyledons and leaves of each host that had been lightly dusted with Carborundum. Transmissions by aphids, Myzus persicae Sulz., were effected in the 2- to 4-true leaf stage in insect proof cages. Virus-free aphids were allowed 30 minutes' virus acquisition time on infected tissue and were then allowed to feed for 2 hours on healthy test plants.

Diagnostic hosts used in these studies were Nicotiana glutinosa, N. tabacum (tobacco "Samsun"), Citrullus vulgaris

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(watermelon "Charleston Gray"), Cucurbita pepo (squash "Early Prolific Straightneck"), Cucumis melo var. cantalupensis (cantaloup "PMR 45"), and Chenopodium amaranticolor.

Greenhouse temperatures were maintained between 65° and 85° F. An insecticide was applied weekly for insect control.

RESULTS AND DISCUSSION

Inoculation of the 12 specimens from La Jolla to a series of diagnostic hosts indicated 11 of the specimens to be infected with WMV and or SMV and a virus inducing necrotic lesions on Chenopodium amaranticolor. Cross-protection tests and host-range studies indicated that the unknown virus was not a closely related strain of cucumber mosaic virus (CMV). Extensive inoculation tests indicate that C. amaranticolor is apparently immune from WMV and SMV, therefore a pure source of the unknown virus by passage of the virus mixture through this host was obtained for further study.

Symptoms on C. amaranticolor: Small pin-point light brown spots with chlorotic borders (Fig. 1) develop on inoculated leaves 6 to 8 days after mechanical inoculation depending on the temperature. At 85° F initial symptoms were discernible 6 days after inoculation and at 70° after 8 days. Cloudy conditions tend to delay onset of initial symptoms and their subsequent development. The small necrotic spots and chlorotic borders enlarge slowly and a red border develops in the chlorotic area around the enlarging necrotic center. After 10 to 12 days most of the lesions reach their maximum size (2 to 3 mm in diameter) (Fig. 2). Lesions developing across or adjacent to veins may develop slight necrosis and red coloration 10 to 20 mm along

FIGURE 2. C. amaranticolor leaf 12 days after inoculation with the latent virus.

FIGURE 3. Pinto bean leaf showing veinclearing 15 days after inoculation with the latent virus.

the veins. Enlarging lesions may coalesce and form large, medium-brown, necrotic spots on inoculated leaves. Affected leaves develop large chlorotic areas along or between veins and usually abscise 15 to 20 days after inoculation. Similar symptoms develop in this host from aphid inoculation at points of feeding. The virus does not become systemic in this host either from mechanical- or aphid-inoculation.

Mechanical and Aphid Transmission: The virus latent in some cucurbits was transferred free of other known viruses from C. amaranticolor and maintained in cantaloup, squash, and watermelon. Transmission from host to host was easily accomplished with inoculum prepared

by grinding young infected leaves in a 0.5 % phosphate buffer (pH 7.8) with a mortar and pestle. Titer of the virus dropped sharply in older leaves of infected plants in the greenhouse. Defoliation or removal of old leaves and forcing new growth by fertilization reestablished the virus in high concentration in new growth of old plants. The virus appeared to remain in high titer in old leaves of spinach, but was not recoverable from inoculated leaves of C. amaranticolor after 18 days.

The virus was readily transmitted by M. persicae from squash to squash, watermelon, cantaloup, garden pea, Pinto bean, spinach, and C. amaranticolor. Preliminary tests indicate that the virus is not persistent in the aphid M. persicae.

Physical Properties: Young infected squash plants were used as the source of inoculum and C. amaranticolor and squash as the test plants in these studies. The results of 3 separate tests are as follows: The virus was infectious after 10 minutes at 50°C but not at 55°, at dilutions including 1:100 but not 1:1000, after storage at 22° for 3 days but not for 4.

Host-range Studies: The virus was infectious to but latent in PMR 45 cantaloup, Charleston Gray watermelon, Marketer cucumber, Summer Straightneck squash, and Dixie Market spinach. Bean variety Pinto and Laxton Progress garden pea were also susceptible to infection by both mechanical means and aphid inoculation. Infected garden pea plants developed a distinct mottle in the young leaves 8 to 10 days after inoculation. Affected plants were only slightly stunted. The distinct mottle in the young leaves was evident throughout the life of the plant. Older leaves were moderately chlorotic. Inoculated plants of bean variety Pinto developed a brilliant yellow veinclearing 12 to 15 days after inoculation (Fig. 3). Infection by the latent virus was verified by inoculation to C. amaranticolor. Slight petiole epinasty developed on affected bean plants. Symptoms became latent with increasing temperatures.

The virus did not induce symptoms in and the virus could not be recovered from inoculated plants of the following: Nicotiana glutinosa, N. tabacum ("Samsun"), N. clevelandii, Gomphrena globosa, Capsicum annuum ("California Wonder"), Beta vulgaris (sugar and garden beet), Datura stramonium, Vigna sinensis ("Black") and Lactuca sativa.

Relationship to Other Viruses: Cross-protection tests indicate no close relationship between the latent virus and tobacco ringspot virus (TRSV), CMV, WMV, and SMV. Each of the viruses inoculated to squash, cantaloup, or cucumber previously infected with the latent virus induced symptoms characteristic of the second inoculated virus. Symptom development or intensity in these hosts did not appear to be materially affected by the presence of the latent virus in the greenhouse. Titer of each virus in dual infections appeared to be similar to that of a single infection by each virus in the same host.

Differences in host range further indicate a lack of relationship between the latent virus and CMV, SMV, TRSV, and WMV. Also, cross-protection and host-range studies indicate no close relationship between the latent virus and sugar beet mosaic virus and lettuce mosaic virus.

Though no direct comparative studies have been made, host-range differences indicate no close relationship between the cucurbit latent virus and the latent virus of California artichokes (2) or the latent virus of dodder (1).

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PHYSIOLOGIC RACES OF PUCCINIA GRAMINIS TRITICI IN SOUTHERN KOREA

Hoo Sup Chung and Bong Koo Chung[1]

The wheat stem rust incited by Puccinia graminis tritici that occurs late in the growing season is known to be a limiting factor of wheat production in Korea because of the serious losses in the yield and quality of the crops. From observations thus far made, there are no susceptible species of barberry of any importance in Korea (1). Control by the eradication of the alternate host, therefore, is out of the question. Although dusting with sulfur has been demonstrated experimentally to control stem rust in other countries, it is not feasible as a practical control measure in Korea. Apparently the only possible practical control measure will be by growing resistant varieties. Consequently, investigation of the physiologic races of Puccinia graminis tritici occurring in Korea is important as a basis of information for the resistant varieties to stem rust of wheat.

Between May and August 1960 wheat infected with the uredial stage of rust was collected in various localities in southern Korea and used for making inoculations on 12 differential varieties listed by Stakman, et al. (2). The usual methods were followed in inoculating, incubating, and so forth. The reactions of the standard varieties to the races were tested twice at temperatures ranging from 18° to 27° C. The results are summarized in Table 1.

Table 1. Reaction of differential varieties of wheat to physiologic races of Puccinia graminis tritici in southern Korea.

Place of collection	Infection type												Physiologic race
	L. C.	Ma.	Rel.	Ko.	Arn.	Mnd.	Spm.	Kub.	Ac.	Enk.	Ver.	Kpl.	
Suwon	4	4	0;	3	4	4	4	3	3	3	1	1	17
Anyang	4	4	0	3	4	4	4	4	3	1	0;	1	21
Taejon	4	4	0	3	4	4	4	4	3	1	0;	1	21
Kwangju	4	4	4	4	4	4	4	4	3	2	0	1	34
Jinju	4	4	4	4	4	4	4	4	4	2	4	2	40
Chungju	3	1	0	0;	0;	0;	0;	0;	0;	0	1	1	111
Yesan	4	4	0	3	4	4	4	0;	0;	0;	0;	0;	194
Chochiwon	4	2	3	3	2	4	2	3	2	1	1	2	K_1
Cheju	4	X	2	2	3	1	0	0	3	1	2	2	K_2

It will be noted from the data presented in Table 1 that the stem rust fungus consisted of many physiologic races in southern Korea. Seven isolates of the fungus identified were namely one each of 17, 34, 40, 111, 194 and two isolates of 21 inclusive. The remaining two isolates among nine collections were not identical with the keys issued by Stakman, et al. (2, 3). It was therefore recognized that two isolates have never before been described and are considered to be entirely new. The two new races were tentatively named K_1 and K_2 inclusive.

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EFFECTIVENESS OF DORMANT SPRAYS IN THE CONTROL OF DUTCH ELM DISEASE

George E. Hafstad and John F. Reynolds

A dormant DDT spray has long been recommended to protect elms from Dutch elm disease. Recently, methoxychlor has also been recommended for spring application, as it has been found to be much less toxic to robins than DDT. Many individuals and some municipalities claim that present recommended sprays for the control of Dutch elm disease are ineffective, even worthless, and that it is impossible for spray equipment to reach the tops of elms 50 feet or more in height.

In order to determine the effectiveness of dormant sprays in protecting elms against beetle feeding, the following test was made. Elms 50 to 80 feet in height were selected at random in different municipalities that had practiced dormant spraying for the control of Dutch elm disease. Five branches were selected from five different places in the uppermost parts of the trees. From each branch, five twigs were taken, each with five twig crotches. These twigs were then placed in five different, new, screened gallon cardboard containers. Ten smaller European elm bark beetles, Scolytus multistriatus, less than 24 hours old, and which had had no opportunity to feed, were placed in each container. After 24 hours the containers were checked, and the beetles classified and counted as dead, dying, or alive.

A check, or control, was included for each series. This consisted of five twigs with five twig crotches from a non-sprayed tree. These were placed in the same type container, and ten smaller European elm bark beetles of the same age and from the same lot as those used for the sprayed trees were placed in the container. After 24 hours the container was checked, and the beetles were classified and counted as dead, dying, or alive.

The purpose of the dormant spray is to prevent beetles from feeding on healthy elms and thus causing possible infection. If feeding occurs before the beetles succumb, the spray is of little value. It has been determined through research investigations at the University of Wisconsin that feeding niches three or more millimeters in length are sufficient to cause infection[1]. Because of this fact, every twig was carefully examined with a hand lens after being exposed to beetles for a 24-hour interval for evidence of beetle feeding. All niches found were measured by means of a wire gauge.

A total of 18 different sprayed trees from 11 cities were tested in the described manner. These trees included those having had either a fall or a spring application by a mist blower or a hydraulic sprayer with either DDT or methoxychlor dormant sprays. A total of 914 smaller European elm bark beetles were used on the sprayed twigs. After 24 hours, 708 were dead, 191 dying, and 15 were alive. On the 450 twigs used, nine feeding niches were found, varying from 1 to 4 mm, with an average length of 2.3 mm.

Twelve trees were used as checks as previously described. None of these were sprayed. A total of 114 smaller European elm bark beetles were used in these tests; 24 hours later 101 were alive, 6 were dead, and 7 were dying. Again, every twig was examined with a hand lens after exposure to 24 hours of beetle feeding. On the 60 twigs used, 75 feeding niches were noted, varying from 1 to 5 mm with an average length of 2.8 mm.

These tests are not conclusive, but it is believed that they are sufficient to indicate that excellent control can be obtained by dormant spraying to prevent feeding by smaller European elm bark beetles and subsequent infection. All the trees tested were picked at random from sprayed areas, the only criterion used being that they were trees at least 50 feet in height. Apparently these data refute the statements that sprays are worthless, that spray equipment does not reach the tops of tall trees, or that beetles are becoming resistant to sprays.

In summary, of the 914 smaller European elm bark beetles placed on sprayed twigs, 899 were dead or dying and 15 were alive after 24 hours. Of the 114 smaller European elm bark beetles placed on non-sprayed twigs, 101 were alive, active, and feeding; only 13 were dead or dying.

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INDUCTION OF WOODY GALLS BY WOUNDING OF CITRUS INFECTED WITH VEIN-ENATION VIRUS

James M. Wallace and R. J. Drake[1]

Abstract

Naturally developing woody galls on trees of Rough lemon and sour lime infected with vein-enation virus often arise at the base of thorns. On plants infected by means of tissue grafts, the galls frequently develop at or near the site of inoculation. These findings indicated that gall initiation is associated with tissue wounds. Experimental studies showed that citrus woody galls are induced by a combined action of virus and wounds. It was not determined if galls are initiated in the absence of wounds.

INTRODUCTION

The vein-enation virus disease of citrus was first described by Wallace and Drake (9) in 1953 in California. These authors (10) reported later that the green peach aphid, Aphis persicae (Sulz.) is a vector of the causal virus[2]. In 1954 McClean (8) published results of studies on vein-enation in South Africa where he showed the tropical citrus aphid, Toxoptera citricidus (Kirk.), to be a vector. Fraser (4) in 1958 stated "it (vein enation) appears to be widespread in New South Wales but of no discernible importance." Up to that time studies in California and South Africa had likewise indicated that this disease was probably of minor importance inasmuch as infected trees displayed no injurious effects. In California the writers (10) considered the possibility that the vein enations could be symptoms of some of the citrus virus diseases for which no leaf symptoms were known. A further consideration was a possible relationship between the vein-enation virus and certain bud-union disorders of undetermined cause.

In 1959 Fraser (5) described "woody gall" of citrus in New South Wales which was found to be bud-transmissible and was presumed to be a virus disease. During a visit to California in 1957 Dr. Fraser had observed galls on trees of Rough lemon in the experimental plantings at the University of California, Los Angeles, which she stated were identical with those occurring in New South Wales. Soon thereafter, in correspondence with the senior author of this paper, Dr. Fraser suggested a possible relationship between woody gall and vein enation. This led to studies which gave evidence that the woody gall and vein-enation symptoms are caused by the same virus (11). This has been substantiated by further experiments and, additionally, it has been discovered that the woody galls are induced commonly by a combined action of wounds and virus. The response of infected plants to wounding is very similar to that described by Black and co-workers (1, 2, 3, 6, 7) in their studies of the wound-tumor virus of sweet clover and other hosts.

SYMPTOMATOLOGY

Vein Enations: Symptoms on leaves range from veinal swellings to conspicuous projections on the under surfaces (Fig. 1A). On the upper surfaces shallow indentations correspond to the opposite prominent peg-like structures (Fig. 1B). To the unaided eye and to touch, these veinal outgrowths or papillae seem to be rather sharply pointed. Magnified views (Figs. 1C and 1D) of the large enations show that some are knobby or cup-shaped, gall-like structures. Perhaps they differ from the galls that develop on the woody parts of the plants because they originate on a different kind of tissue. Maturation of the leaves probably stops their development and limits the size they can attain.

Woody Galls: On semi-hard or woody stems, the first sign of gall development is a slight bump or swelling covered with normal-appearing bark. Removal of the bark reveals that the swelling has resulted from an abnormal enlargement of the xylem tissues at that location. As the galls increase in size they gradually attain an irregular shape, and the bark covering the gall tissue becomes slightly rough and lighter in color than normal. In advanced stages, adjacent galls may coalesce. On old, large galls it appears that new galls have continued to originate within the gall tissue producing the "cauliflower-like structures" described by Fraser (4). Figures 2A and 2B show naturally developing woody galls on a field tree of Rough lemon and earlier stage galls on an experimentally infected seedling of the same species.

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[2]L. G. Weathers and E. F. Laird, University of California, Riverside, California, have recently found that Aphis gossypii Glover is also a vector of vein-enation virus. (Unpublished)

The anatomy of the woody galls has not yet been fully investigated and will not be discussed in detail in this paper. As mentioned by Fraser (5), many of the galls appear to have been initiated at an early stage of growth. Commonly (Fig. 3) the abnormal tissue extends in a fan or cone shape from a point of origin near the center of the stem. However, galls can initiate from the cambial zone of older stems.

RELATION OF WOUNDS TO GALL DEVELOPMENT

Early in these studies it was observed that woody galls that developed spontaneously on experimentally infected seedlings commonly were located in close proximity to the thorns. Also, galls appeared frequently in and around the inoculation sites where virus-bearing buds or small twig grafts had been inserted. Thus it appeared that both natural growth wounds and mechanical tissue injuries initiated galls. Experimental tests demonstrated that wounding of vein-enation-infected citrus plants induced woody galls on stems and on roots.

Wounding of Trunk and Stem Tissues: Punctures by means of a small insect pin or a sharp-pointed dissecting scalpel were made in diseased and healthy plants of Rough lemon and sour lime. On the hardened woody stems the wound extended through the bark to the outer xylem tissues. On very small, young twigs the needle punctures frequently reached or penetrated the pith region. Preliminary anatomical studies of relatively deep punctures of soft stems indicated that the initial response of virus-infected tissues to wounding was similar to, or the same as, that of healthy plants. In both a considerable amount of abnormal tissue developed in the pith (if injured), the internal xylem, and cortex. As a result of the healing processes around the punctures, there were at times slight external swellings on the healthy control plants. These closely resembled the first detectable stage of true galls that develop with or without wounding on infected plants.

Subsequent examination disclosed that the slight elevations or "bumps" on the bark of punctured healthy plants were normal responses to wounding since they did not develop further. Sections showed no continuity between the areas of abnormal tissue in or near the pith and those in the bark at the point of injury. Sections through early stage wound-induced galls on infected plants sometimes revealed continuous bands of abnormal tissue extending from deep within the xylem to the outer surface, and there was more bark tissue proliferation around the wound than was present on the healthy control plants.

Figure 4A shows gall development 4 1/2 months after punctures were made into the woody stem of a Rough lemon seedling infected with vein-enation virus. A similarly wounded healthy plant (Fig. 4B) had only slight wound scars at the sites of the punctures.

Table 1 summarizes the results of one experiment on the relation of wounds to gall development. Counts were made 4 1/2 to 5 months after wounding. By that time 63% of the wounds on vein-enation-infected plants had resulted in woody gall development. No galls had developed on wounded healthy control plants.

Table 1. Wound-induced woody galls on young seedling trees of Rough lemon and lime.

Variety	Number of plants	Ratio (galls/wounds)
Rough lemon (I)[a]	4	71/94
do. (H)[b]	2	0/97
Mexican lime (I)[a]	4	76/139
do. (H)[b]	2	0/129

[a]Infected with vein-enation virus.
[b]Healthy controls.

Wounding of Roots: Examination of roots of potted plants revealed some galls on roots of infected plants but none on roots of healthy plants. Infected and healthy lime plants were grown in water culture to facilitate studies of gall development on roots. When roots of diseased plants were wounded by means of pin punctures, galls developed at most of the wound sites. However, in water culture, many galls developed where no experimental wounds were made. There was a suggestion that wounding stimulated gall development at locations distant from the wounds, but this has not been determined with certainty. Gall development on roots is shown in Figure 5.

On healthy plants wounding resulted only in wound scars. Figure 6 pictures the reactions of punctured roots of infected and healthy plants. In the studies of gall development on roots, a much greater number of galls have developed on non-injured roots of diseased plants grown in water culture than on plants maintained in soil or sand cultures. In fact, so many galls developed spontaneously over the root system of water culture plants that after 2 to 3 months it was difficult to identify those that developed from experimental punctures.

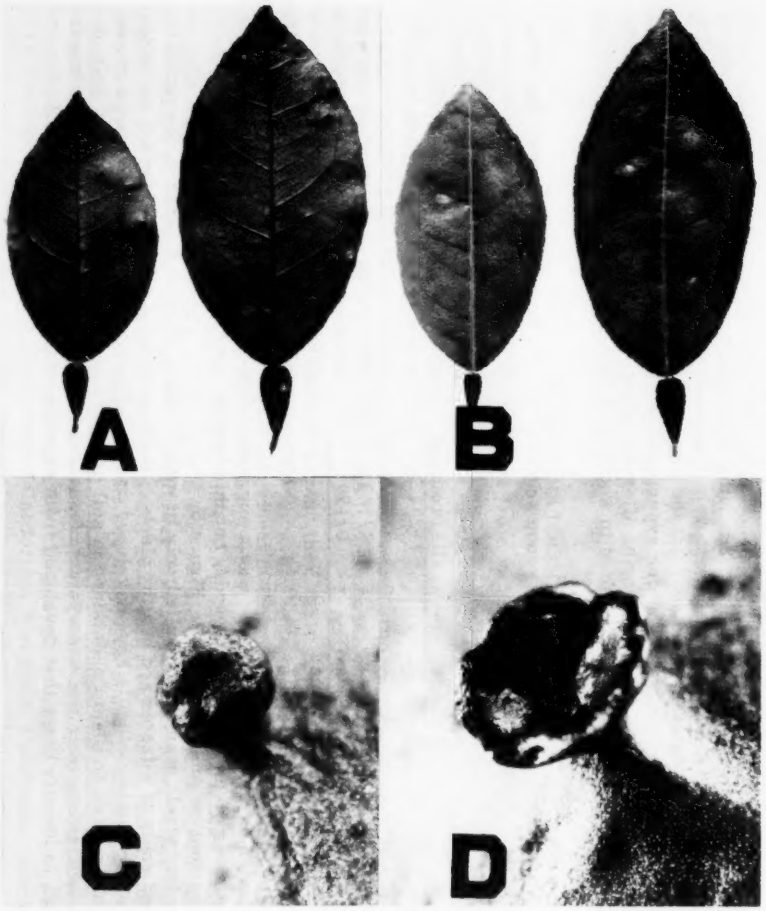


FIGURE 1. Vein enations on leaves of Mexican lime. A -- Enations and swollen veins on under surface of leaves. B -- Indentations on upper surface corresponding to enations. C and D -- Magnifications of individual enations.

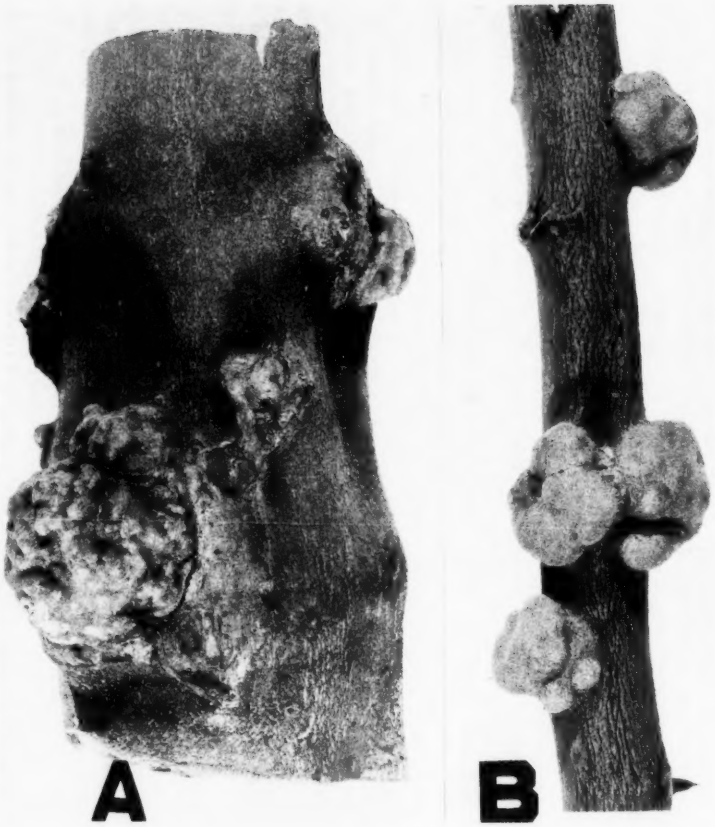
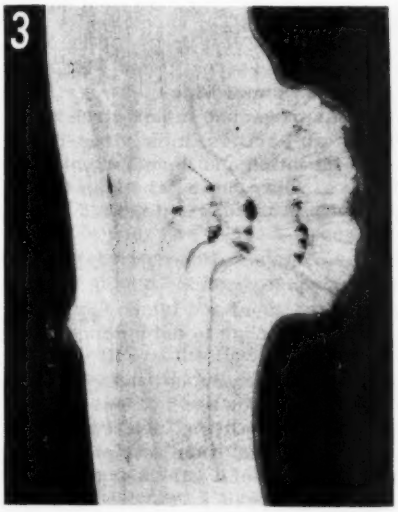
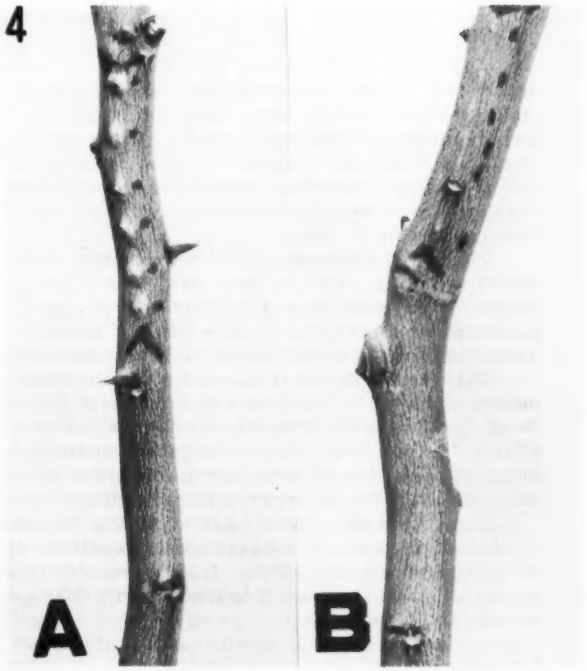


FIGURE 2. Woody galls on citrus. A -- Naturally-occurring galls on field tree of Rough lemon. B -- Galls on Rough lemon seedling experimentally infected with vein-enation virus.





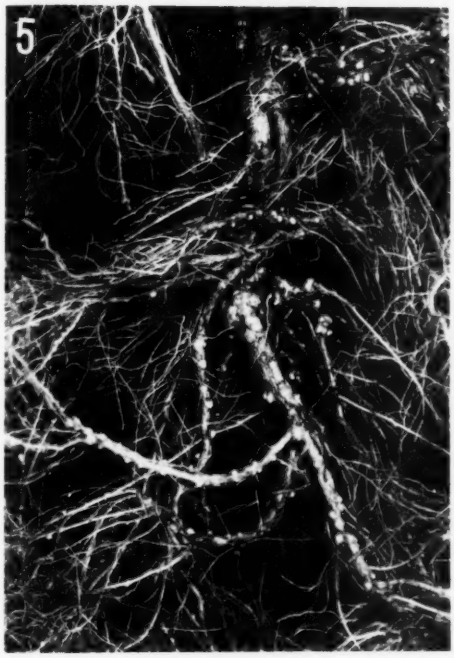


FIGURE 3. Section through affected stem showing cone of gall tissue extending from near the center of the woody cylinder. Dark areas are gum deposits.

FIGURE 4. Wound-induced woody galls. A -- Early stage galls developing at the sites of 6 pin punctures of a lime seedling infected with vein-enation virus. B -- Slight wound scars at sites of punctures of healthy plant. Black dots were for the purpose of identifying punctures.

FIGURE 5. Gall development on roots of virus-infected lime plant grown in water culture 6 months. At beginning of experiment wounds were made in the tap root and some laterals. Galls began to develop at the wounds but later could not be identified because of general development of galls at sites where no experimental wounds were made.

FIGURE 6. Wound response of roots of diseased and healthy plants grown in water culture. Left -- Two diseased roots with tumorous galls at most of the experimental wound sites. Right -- Two roots from healthy plant showing only wound scars. Photographed 6 months after wounds were made.

DISCUSSION

The evidence that woody gall of citrus is caused by the vein-enation virus is now well substantiated. It has been established also that vein-enation is a wound tumor virus. This virus and its effects on host plants have not been investigated as fully as the wound tumor virus of sweet clover and other host plants. There are, however, many similarities in the virus-host reactions of the two viruses. Anatomical studies of woody gall initiation and development are just beginning. As in the case of the clover wound tumor disease, there are no clues as to the exact nature of the combined action of wound and virus that results in the development of the tumorous galls on citrus.

The writers have not found vein-enation woody galls on sour orange and commercial sour lemon varieties. Both of these are hosts of the virus, and sour orange develops strong leaf enations. Stem punctures of infected sour orange (California standard sour) and lemon (Eureka) seedlings have not induced galls within 4 months. No tests have been made to determine if wounding of roots of these species will stimulate gall development.

Old trees of Rough lemon and other varieties growing on Rough lemon rootstocks in experimental plantings in California do not appear to be injured even though galls are present on the Rough lemon parts. Possibly such trees had attained a large size before being infected. The effects of early infection of susceptible varieties such as Rough lemon and lime have not been determined. Neither of these species is grown commercially in the parts of California where the vein-enation virus is known to be spreading naturally.

Regardless of economic potentialities, the vein-enation virus has been of interest to plant virologists because of the synergistic reactions demonstrated by Weathers (12) between it and the citrus yellow-vein virus. It becomes still more interesting with the discovery that it has the somewhat unusual capacity to cause tumor-like galls which originate from a combined action of wounds and virus.

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THE IMPORTANCE OF GREEN RING MOTTLE VIRUS DETECTION IN A PRUNUS VIRUS INDEXING PROGRAM[1]

P. R. Fridlund[2]

Abstract

The green ring mottle virus of Prunus was very commonly found in select clones of symptomless Prunus avium, sweet and mazzard cherry, and P. avium x P. cerasus, duke cherry. Natural infections in symptomless Prunus persica, peach, and Prunus armeniaca, apricot, also occur. The frequency of infected clones warrants indexing for this virus in any reliable indexing program.

Indexing of the Prunus clones accepted for introduction into the Interregional Fruit Tree Repository, (IR-2), has shown a number to be infected with viruses. Although it is suggested that the clones should be fairly well indexed for virus before they are proposed for the repository, admittedly many are not. Thus the clones received have had a variable range in previous indexing, from none at all to very thorough on a number of indicators.

When received by the IR-2 Project, the clones are screened for viruses on Shiro-fugen flowering cherry, Prunus serrulata, and later on National Pickling cucumber, Cucumis sativus, to detect the commonly occurring ring spots, cherry yellows, prune dwarf, and similar latent virus strains. In bud inoculated Shiro-fugen (4, 6) these latents cause a hypersensitive reaction expressed as a localized necrosis around the inserted bud, usually with gumming. In juice inoculated cucumber (2, 7) the latents cause chlorotic spotting of the cotyledons, systemic leaf patterns, or death of the host. If any clones are found to be virus-infected during this preliminary screening, they are discarded immediately with no further indexing. If no virus is detected, the clones are then indexed thoroughly on a standard series of ten Prunus varieties which will detect practically all known Prunus viruses.

Some time after the IR-2 indexing program began, Prunus avium clones were found to contain the green ring mottle virus in unusual frequency. Consequently, P. avium and duke cherry, P. avium x P. cerasus were also screened on Kwanzan flowering cherry, P. serrulata, to detect the green ring mottle virus. The green ring mottle virus causes an epinasty symptom in bud inoculated Kwanzan (1, 3, 5) and to a milder extent in Shiro-fugen in the absence of ring spot. Ringspots do not cause local necrosis in Kwanzan.

Seventy-five of 363 Prunus clones, for which indexing has been completed or terminated, were found to be virus infected. The disease pattern is shown in Table 1. As some clones contained multiple infections, more viruses than infected clones are indicated. Since all clones of indexed P. serrulata were either Shiro-fugen or Kwanzan, and as no little cherry virus-free clones were then known, universal infection was more or less anticipated.

The frequent occurrence of the green ring mottle virus as shown was not anticipated. Apparently it is more prevalent and has a wider natural host distribution than previously realized. It is probable that it occurs even more frequently than shown because under earlier indexing procedures, ring-spot infected clones were discarded immediately upon detection before any indexing for the green ring mottle virus was done. Previously, the occurrence of this virus had been considered rare (8), although a later report (5) states it to be common in the western United States.

In a supporting exploratory experiment, the indexing of 82 latent virus-free clones of miscellaneous varieties of sweet cherry, P. avium; sour cherry, P. cerasus; duke cherry, P. avium x P. cerasus; and P. mahaleb, mahaleb cherry, substantiated these findings as shown in Table 2.

Green ring mottle is often found in P. avium and P. avium x P. cerasus as shown by the data. It is also common knowledge that green ring mottle-free clones of certain varieties such as Lambert, Napoleon (Royal Ann), and Deacon are almost impossible to obtain. Since the virus is symptomless or nearly so in these species, any reliable indexing program must contain Kwanzan or Montmorency, P. cerasus, to detect this virus.

[1]Scientific paper No. 2132, Washington Agricultural Experiment Stations, Pullman, Washington. This work was conducted under Project No. 1262 and was supported by Interregional Research Project IR-2.

[2]Associate Plant Pathologist and Plant Pathologist in charge, IR-2, Department of Plant Pathology, Washington State University, Irrigation Experiment Station, Prosser, Washington.

Table 1. The occurrence of viruses in Prunus clones received for introduction into the Interregional Fruit Tree Repository (IR-2).

Species of Prunus	Number of clones: indexed	Number of clones: infected	Viruses found: ringspot, cherry yellows, prune dwarf, etc.	Viruses found: green ring mottle	Viruses found: others
P. serrulata, flowering cherry	8	8	1	-	8
P. avium, sweet cherry and mazzard	86	41	28	17	2
P. cerasus, sour cherry	27	2	2	-	-
P. avium x P. cerasus, duke cherry	3	2	-	2	-
P. mahaleb, mahaleb cherry	26	0	-	-	-
P. persica, peach and nectarine	82	12	6	6	-
P. armeniaca, apricot	20	3	2	1	-
Prunus species, various plums	76	7	7	-	-
Prunus species, other species	35	0	-	-	-
Totals	363	75	46	26	10
				82	

Table 2. The occurrence of green ring mottle virus in miscellaneous cherry clones which were free from the ringspot, cherry yellows, prune dwarf and similar latent viruses.

Species of cherry	Number of clones: indexed	Number of clones: infected
P. avium, sweet cherry and mazzard	66	9
P. cerasus, sour cherry	10	0
P. avium x P. cerasus, duke cherry	3	1
P. mahaleb, mahaleb cherry	3	0
Totals	82	10

This virus disease is clearly expressed as green spots, rings, or arcs on P. cerasus foliage (8), and this probably accounts for its absence in the clones received for the repository. Budwood from symptom-expressing trees would naturally not be forwarded.

Although the virus is apparently symptomless in P. mahaleb, no infected trees were found. Other data (3) substantiate these results. Apparently there are very few if any naturally infected mahalebs.

The six natural infections of P. persica and one of P. armeniaca as shown in Table 1 were diagnosed as green ring mottle on the basis of a mild epinasty in Kwanzan and Shiro-fugen. This epinasty differed from that obtained from cherries only in that it was always much milder.

Circumstantial generalities regarding the diseases received in clones also indicate a definite disease pattern. If the clones are arbitrarily divided into groups of no previous indexing, some indexing, and thorough indexing, in general the following pattern appears: Little virus was found in thoroughly indexed material, and that which occurred could be accounted for through recent field spread. Ringspots and allied latents were by far the most prevalent in the nonindexed or little-indexed material, indicating that in a partial indexing program these are the first viruses selected for detection. This is reasonable as they are both the most frequent and the easiest to detect through indexing. However, green ring mottle occurred more or less uniformly between non-indexed and partially indexed clones.

These data emphasize the false security obtained in a partial indexing program. It is

true that the commonest infective agents, the ringspots and allied latents, are usually eliminated with a partial program, but other symptomless viruses may be very common and are readily overlooked as shown here for the green ring mottle virus.

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A NEW RACE OF BEAN RUST IN ARKANSAS

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Abstract

Race 34, a new physiologic race of bean rust (Uromyces phaseoli var. typica Arth.), has caused serious damage to bush snap beans in Arkansas. Epidemics occurred during the late summer of 1959 and 1960. Most varieties of bush snap beans tested are susceptible. The known distribution extends from the Arkansas river valley northward through the Ozark Mountains in northwest Arkansas. Race 34 is similar to race 32 in that it is highly infectious to bush snap beans.

During the late summer and fall seasons of 1959 and 1960 the commercial bush snap bean (Phaseolus vulgaris) crop in northwest Arkansas was heavily damaged by rust. The disease became epidemic throughout the area, and numerous fields were abandoned as plants became defoliated.

A study was made to determine if a new physiologic race of the bean rust fungus was associated with the epidemic. Spores were collected from infected leaves of the variety Top Crop. During the winter of 1959-60 different bean varieties were inoculated in the greenhouse. Susceptible varieties included Top Crop (Fig. 1), Contender, Tenderbest, Bountiful, Dwarf Horticultural, Landreths Stringless Green Pod, Commodore Improved, Bachicha, Ferry's Plentiful, Processor, Wade, Sulphur, Giant Stringless Green Pod and Tender Green. The variety Tennessee Green Pod was immune. Monarch blackeye pea was also immune.

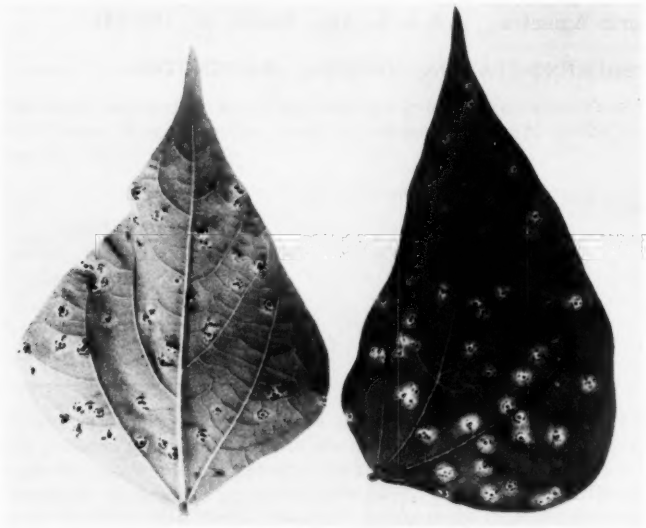

FIGURE 1. Reaction of Top Crop to bean rust race 34.

In July 1960 a new pathogenic race of the bean rust fungus was reported from Maryland[1]. This race was reportedly pathogenic to most bush green bean varieties and non-pathogenic to several of the pole varieties including Kentucky Wonder and several strains of Blue Lake. Spores from the Arkansas rust isolate were sent to Beltsville for comparison with the newly described race 32. Dr. W. J. Zaumeyer compared the two isolates by studying their relative virulence to the standard differential bean varieties. His results are presented in Table 1.

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[1] Zaumeyer, W. J. 1960. A new race of bean rust in Maryland. Plant Disease Reptr. 44:459-462.

Table 1. Reaction of the differential bean varieties to the Arkansas race and race 32.

Differential variety	Infection grade[a] produced by rust race	
	Ark. race	Race 32
U.S. No. 3	2	1/3[b]
Bountiful	10	10
No. 643	1	0
Pinto	4-5	0
No. 765	1	0
No. 780	1	1
No. 814	0	0
Golden Gate Wax	8	3/10
Top Crop	10	10

[a]Infection grade 10 indicates the most severe reaction and 0 indicates immunity.

[b]Numerator indicates size of pustule on upper leaf surface and denominator on lower surface.

The Arkansas race differs from race 32 in its reaction to Pinto and Golden Gate Wax. It also differs from any other race previously reported. The Arkansas race is designated race 34.

The known distribution of race 34 extends from the Arkansas river valley northward through the Ozark Mountains in northwest Arkansas.

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RESPONSES OF TOBACCO BREEDING LINES TO THREE SPECIES OF ROOT-KNOT NEMATODES IN GREENHOUSE TESTS

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Summary

Selected tobacco breeding lines were consistently resistant to the common root-knot species, Meloidogyne incognita acrita, in greenhouse tests but were susceptible to M. javanica and M. arenaria. The last two species are infrequently found in tobacco in the North Carolina, South Carolina, and Georgia tobacco areas. Possible further spread of the less common root-knot species in the tobacco areas indicates a need for plant breeders to develop a wider range of resistance that would include other root-knot species.

INTRODUCTION

The tobacco breeding line PD 611, which is resistant to the root-knot nematode Meloidogyne incognita acrita, was released in 1959 to plant breeders for use as a parent source of nematode resistance (4). This release illustrates the usefulness of a new source of root-knot resistance originated by Clayton, et al. (1) from the Nicotiana alloploid N. sylvestris x N. tomentosiformis and the tobacco introduction TI 706. The commonly occurring root-knot nematode species on tobacco in North Carolina and South Carolina is M. incognita acrita as reported by Clayton, et al. (1). Additional species determinations by the writer confirmed these observations. Field nursery test plots at Florence, South Carolina which were used in the breeding program to develop tobacco varieties resistant to root knot were infested only with M. incognita acrita. Numerous breeding lines now show high resistance to this species.

When certain selected breeding lines showing high resistance in North Carolina and South Carolina were grown in nematode-infested nursery plots at Gainesville, Florida and Tifton, Georgia, varying degrees of root-knot infection were observed. Root-knot species determinations from root collections at Gainesville and Tifton revealed that M. javanica and M. arenaria along with M. incognita acrita were present in mixed populations. The variable response of breeding lines grown in soil infested with mixed species of root-knot nematodes was of special interest because of the possible future need for a wider range of resistance in tobacco to more than one species. This report presents data on: 1) root-knot nematode species found in Georgia and Florida nursery breeding plots and 2) on the response of selected tobacco breeding lines to three species in greenhouse soil cultures at Florence, South Carolina.

GEORGIA AND FLORIDA COLLECTIONS

Tobacco root samples were collected at Tifton and Gainesville in 1958 and in 1959, and in Tifton only in 1960. Most of the root samples were taken in nursery breeding plots that had a history of severe root-knot infestation. In plots at Gainesville tobacco was grown continuously, and at Tifton it was grown in a 2-year rotation with corn.

The identity of root-knot species was determined from individual mature female specimens. Cuticle perineal patterns from specimens were mounted in Lacto-phenol and examined with an oil-immersion lens. The method of mounting was the same as that used by Taylor, et al. (3). Results (Table 1) from the Tifton collections show that M. incognita acrita was most numerous in 1958 as it was present in 11 of 14 collections. In 1959 and 1960 however M. javanica was found with about equal frequency as M. incognita acrita. These two species were therefore variable in occurrence over the 3-year period and neither of them was consistently dominant. In several cases two or more root-knot species were found in a single collection including M. arenaria in addition to those just named. From eight collections at Gainesville in 1958, four were determined as M. incognita acrita, three as M. javanica, and one a mixture of four species (Table 1). In 1959 four collections at Gainesville contained only M. javanica.

Table 2 presents data obtained in 1959 after examining selected nematode-resistant breeding lines that were planted in plots at each of three locations (Florence, Tifton, and Gainesville).

[1]Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture and South Carolina Agricultural Experiment Station, Florence, S. C.

Table 1. Root-knot species identified from tobacco root collections[a] at Tifton, Georgia and Gainesville, Florida during 1958, 1959, and 1960.

Location and root-knot species	Year and number of collections in		
	1958	1959	1960
Tifton, Georgia:			
Total number collections	14	18	22
Meloidogyne javanica	1	8	9
M. incognita acrita	11	8	10
Mixed species[b]	2	2	3
Gainesville, Florida:			
Total number collections	8	4	-
M. javanica	3	4	-
M. incognita acrita	4	0	-
Mixed species[c]	1	0	-

[a]Each collection consisted of galled roots from a single tobacco plant made during August after harvesting was complete. Species determinations were made from perineal pattern examinations of four to ten specimens from each collection.
[b]M. javanica, M. arenaria, and M. incognita acrita.
[c]M. arenaria, M. incognita, M. javanica, and M. incognita acrita.

Table 2. Root-knot development on selected tobacco breeding lines in field nursery plots in South Carolina, Georgia, and Florida in 1959.

Tobacco breeding line	Location and root-knot species		
	Florence, S. C.	Tifton, Ga.	Gainesville, Fla.
	M. incognita acrita[a]	M. javanica[b]	M. javanica[b]
PD 614	none	present	present
PD 615	none	present	present
PD 420	none	present	present
PD 611	none	present	present

[a]Based on examination of two to three single plot rows of 18 to 20 plants each. All plants were free of root knot. Adjacent susceptible varieties were heavily galled.
[b]Based on single plant collections. Severity of galling was slight to moderate. Species identity was determined by perineal examination.

The four entries were selected as root-knot resistant from the nursery breeding plots at Florence, where the species M. incognita acrita only was present. All four entries (Table 2) were free of root knot at Florence but were infested with M. javanica in the Tifton and Gainesville plots[2].

GREENHOUSE EXPERIMENTS

The foregoing field results showing South Carolina selections resistant to local root-knot species and susceptible to those in Georgia and Florida suggested a more critical inquiry by means of greenhouse experiments. Root-knot galls from tobacco were therefore used to establish soil culture of several root-knot species. Cultures were started from single egg masses, each picked from a single isolated mature female. Species identity was confirmed by perineal pattern examination of each nematode specimen. Egg masses were placed in separate crocks of autoclaved soil. Tomatoes were used as host plants as a means of increasing nematode populations.

[2]The writer is indebted to J. G. Gaines, Pathologist, Agricultural Research Service, United States Department of Agriculture, Tifton and Fred Clark, Agronomist, Florida Agricultural Experiment Station, Gainesville for testing these breeding lines in their respective breeding plots and for assistance in collecting and examining root samples.

Tobacco breeding lines that were entered in the first experiment were stabilized in resistance as shown by repeated tests in field nursery plots at Florence. Nematode resistance in these lines originated from the alloploid hybrid developed by Clayton, et al. (1). Separate greenhouse soil cultures of M. incognita acrita and M. javanica were each planted with 10 breeding lines. Tobacco plants grew 8 weeks in infested soil. They were then removed and roots were washed to determine the extent of nematode gall development. Data in Table 3 show that plants grown in soil infested with M. incognita acrita were free of root knot, while the same lines grown in M. javanica infested soil were, with a few exceptions, severely galled. Thus, most of the lines were clearly resistant to the species common in South Carolina and susceptible to M. javanica.

Table 3. Root-knot development on selected tobacco breeding lines grown in soil infested with Meloidogyne javanica and M. incognita acrita in a greenhouse test[a].

Tobacco breeding line	M. javanica soil: Severity of galling[b] (number of plants)					M. incognita acrita soil: Severity of galling (number of plants)				
	0	VSl	Sl	Mod	Sev	0	VSl	Sl	Mod	Sev
112 A	0	0	0	0	2	10	0	0	2	0
208 A	0	0	0	1	1	17	0	0	0	0
307 A	0	0	0	0	2	18	0	0	0	0
308 B	0	0	0	0	2	19	0	0	0	0
608 C	0	0	0	0	2	19	0	0	0	0
608 D	0	0	0	0	2	20	0	0	0	0
609 A	0	0	0	0	2	8	0	0	0	0
Bel 549[c]	1	0	1	0	0	8	0	0	0	0
Bel 633	0	0	2	0	0	13	0	0	0	0
Bel 631 A	12	0	1	0	0	18	0	0	0	0

[a]Although this test was not conducted with an equal number of plants or at exactly the same time, conditions were favorable for root-knot development following massive inoculation dosages. Test was conducted during October to December 1959.

[b]Severity of galling: 0 = none, VSl = very slight, Sl = slight, Mod = moderate, and Sev = severe.

[c]Bel lines 549, 633, and 631 A were kindly supplied for these tests by H. E. Heggestad, located at the Beltsville, Maryland Plant Industry Station, Crops Research Division, where these entries were developed and selected.

A second greenhouse experiment was started in June 1960. Breeding lines with a history similar to those shown in Table 3 were planted in established soil cultures of three root-knot species as indicated in Table 4. Two susceptible tobacco varieties commonly grown in South Carolina, White Gold and Coker 316, were planted as controls. Test entries were planted in heavily infested soil in 4-inch clay pots. After 8 weeks they were removed and roots washed free of soil. The degree of root-knot infection is shown in Table 4 by the severity of galling and the number of egg masses produced. The breeding lines in the soil infested with M. incognita acrita were again free of galls and egg masses, while the two susceptible varieties, Coker 316 and White Gold, were heavily galled and had numerous eggs. The same tobacco lines in soil infested with M. arenaria or with M. javanica were susceptible to these species as galls and eggs were numerous; susceptible control varieties growing in these cultures also were severely galled and had numerous eggs. Galls and eggs in M. javanica were somewhat more numerous than in soil containing M. arenaria. The breeding line Bel 631 in M. javanica soil had only slight galling and very few eggs. Drolsom and Moore (2) reported Bel 631 as susceptible to M. javanica, M. arenaria, and M. hapla. An earlier test by the writer also indicated Bel 631 as susceptible to M. javanica. This line was therefore inconsistent in response to this species.

DISCUSSION

Considerable significance is attached to the demonstration of difference in pathogenicity among the root-knot nematode species on tobacco breeding lines. A desirable objective in further variety development obviously would be a wider range of resistance that would include all the root-knot species. The selections used here were repeatedly grown and tested in soil infested with M. incognita acrita and the present resistance to this species appears to be the re-

Table 4. Severity of galling and egg masses produced on roots of tobacco breeding lines and varieties by three species of root-knot nematodes.

Line number or variety	M. incognita acrita (number of plants)						M. arenaria (number of plants)						M. javanica (number of plants)					
	0[a]	VSl	Sl	Mod	Sev	egg masses	0	VSl	Sl	Mod	Sev	egg masses	0	VSl	Sl	Mod	Sev	egg masses
570 A[b]	6	0	0	0	0	none	0	0	5	0	0	many	0	1	1	3	0	many
570-1	6	0	0	0	0	do.	0	0	4	2	0	do.	-	-	-	-	-	-
570 AB	4	0	0	0	0	do.	0	1	0	1	0	do.	0	0	2	2	1	many
570 B1	6	0	0	0	0	do.	0	0	3	1	0	do.	0	2	3	0	0	do.
603 A	6	0	0	0	0	do.	0	2	4	0	0	do.	0	0	6	0	0	do.
PD 611	6	0	0	0	0	do.	0	0	6	0	0	do.	0	0	3	2	1	do.
Bel 631	6	0	0	0	0	do.	0	0	0	1	0	do.	0	2	2	0	0	few
Bel 633	6	0	0	0	0	do.	0	0	4	0	0	do.	0	1	5	0	0	many
8069-5(NC 95)	6	0	0	0	0	do.	0	0	5	1	0	do.	0	1	3	1	0	do.
White Gold control	0	0	2	2	2	many	-	-	-	-	-	-	0	0	0	0	5	do.
Coker 316 control	0	0	0	2	4	many	0	0	3	2	0	many	0	0	0	3	3	do.

[a]Severity of galling: 0 = none, VSl = very slight, Sl = slight, Mod = moderate, and Sev = severe.
[b]Breeding lines 570 A, 570-1, 570 AB, and 570 B1 were selections by J. G. Gaines at Tifton, Georgia. Line 8069-5 was developed by E. L. Moore at Oxford, N. C. and was released as NC 95 in 1960.

sult of a combination of intensive selection and a favorable source of resistance from the hybrid alloploid and TI 706.

Whether resistance to M. javanica and M. arenaria could also be found in the genotype of the alloploid N. sylvestris x N. tomentosiformis is of interest and is an objective in further study of this source of resistance. Some indication of possible resistance to M. javanica, for example, was shown in the line Bel 631 in one test, but this tobacco now appears to be unstable in this respect. Meloidogyne hapla and M. incognita also are parasitic on the commonly grown tobacco varieties and are being considered in further studies. The information now available indicates that M. incognita acrita is commonly found in the tobacco areas of the Carolinas and Georgia and that other root-knot species are infrequently found on tobacco in the same area. It seems doubtful that the prevalence of M. incognita acrita would indicate a relation to the cropping practices with tobacco in the Carolinas as various crops are used in rotations. In South Carolina cotton is frequently alternated with tobacco, but this practice appears to be less frequent in North Carolina and Georgia. In nursery breeding plots at Florence, a 2-year rotation with corn has maintained populations of M. incognita acrita and the same species has been present in plots of continuous tobacco.

Flue-cured tobacco areas in the southeast are rather strictly limited to suitable soil types and because of this the crop is cultivated rather intensively. Rotation of tobacco with other crops is the general practice and the most suitable land would probably be planted to tobacco in at least 2 of every 5 years. Possibly this cultural practice is more favorable for the perpetuation of M. incognita acrita, or tobacco may be a more suitable host to this species.

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SEPTORIA LEAFSPOT DISEASE OF SCOTCH SPEARMINT

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Two species of spearmint are grown commercially in the midwestern region of the United States for their essential oils. These are common spearmint, Mentha spicata, and Scotch spearmint, M. cardiaca. Some 10,000 acres are devoted to these two species in northern Indiana, southern Michigan and Wisconsin, with a total oil production of 325,000 pounds.

During the current growing season (1961) mint growers in DeKalb, Jasper, Joseph, Kosciusco, Marshall, Noble and Pulaski counties in northern Indiana have reported moderate to heavy incidence of a leafspot disease on Scotch spearmint that has been identified as Septoria leafspot. Green and Sawada[2] reported the occurrence of this disease in 1959 and identified the causal organism as Septoria menthae (Thuem.) Oud. The symptoms of Septoria leafspot disease of spearmint include circular to irregular leaf lesions with dark borders or, infrequently, irregular patches. The leaf lesions are pale green and water-soaked, at first, and turn brown with gray-white centers (Fig. 1). Small, dark colored pycnidia occur within the leaf lesions. When plants are heavily attacked, the defoliation is severe and oil yields are reduced.

FIGURE 1. Foliar symptoms of Septoria leafspot of Scotch spearmint, Mentha cardiaca.

Studies of environmental conditions favorable for infection and disease spread show that periods of 48 to 96 hours of continuous wetting of the foliage are optimum for infection (unpublished data). In the past, this disease could be found in practically every commercial planting of Scotch spearmint in the Midwest but was not considered an important problem. The fact that the disease has appeared in epiphytotic proportions during the present season suggests that environmental conditions are the determining factors for disease development. To date, the season has been characterized by temperatures below normal (2° to 8° F) with slightly above normal rainfall. The cool temperatures have favored heavy dews and prolonged wetting of foliage.

Because this damage has been considered of minor importance in the past, little is known concerning effective control practices.

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SYSTEMIC SPREAD OF TRISTEZA IN ONE VALENCIA ORANGE TREE

Harry C. Burnett[1]

The tristeza virus of citrus is known to be transmitted by Aphis gossypii Glover and Aphis spiraecola Patch in Florida[2]. However, there has been little or no evidence previously reported on the time required for the virus to move throughout the tree after initial infection.

Citrus trees in the Florida Budwood Registration Program are periodically checked for the presence of the tristeza virus. In the course of reindexing some of these trees, one tree, a Valencia sweet orange on sour orange rootstock, was found to be free from the tristeza virus when indexed on Key lime indicator plants in March 1955. However, tristeza was known to be present in the same grove at the time this tree was first tested.

In May 1959, this apparently virus-free tree was reindexed for tristeza, and again there was no evidence of the tristeza virus infection. But, when the tree was reindexed in September 1959, one out of five Key lime test plants gave a positive reaction for the tristeza virus. In December of the same year, budwood was collected at random from different parts of this tree and placed on Key lime test plants. Thirteen plants with live buds were used in this test, but no symptoms of tristeza were observed. Another test was begun in April 1960, and resulted in tristeza symptoms in one of two test plants. In July 1960, another random sample of budwood was collected from this tree. When the indexing was completed, 11 Key lime plants gave a positive indication of tristeza and 8 a negative.

The final test was started on this tree in October 1960. Forty-four sets of budwood were collected from all areas, from the lowest branches to the very top of the tree. Two buds from each stick were placed on seedling Key lime test plants. By January 1961, all Key lime plants with buds from the 44 sets of budwood showed positive symptoms of tristeza. The results of the indexing and reindexing during this 67-month period are shown in Table 1.

Table 1. Results of indexing and reindexing of one Valencia orange tree for the presence of tristeza during a 67-month period.

Test no.	Date budwood collected	No. of test plants	No. positive for tristeza
1	3-9-55	4	0
2	6-1-59	2	0
3	9-2-59	5	1
4	12-21-59	13	0
5	4-11-60	2	1
6	7-12-60	19	11
7	10-17-60	44	44

Assuming that the tristeza virus was transmitted by insects from previously infected trees in the grove to this previously tristeza virus-free tree, the results of this study give some indication as to length of time required for the virus to move throughout the tree following initial infection.

Thirteen and one-half months elapsed from the time the first test plant indicated a positive tristeza reading until all parts of the tree became systemically infected with the virus. If this can be considered as the usual period of time required for a tree to become completely infected, it would be of considerable worth in evaluating tristeza indexing. Normally, four to six sticks of budwood are taken from scattered areas in and around the periphery of the tree. One or two buds are placed in each of four Key lime indicator plants. This procedure is followed each year in the reindexing of each candidate tree. Where an early infection is confined to a small area in the tree, the chances of getting infected budwood may be small, and a tristeza negative reading may result. However, within the following 12-month period, tristeza positive readings can be expected, even when a few buds from the tree are used.

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[2]Norman, P. A., and T. J. Grant. 1956. Transmission of tristeza virus by aphids in Florida. Proc. Florida State Hort. Soc. 69: 38-42.

OBSERVATIONS ON RED CLOVER DISEASES IN MICHIGAN[1]

H. D. Niemczyk[2] and R. L. Kiesling[3]

Abstract

Observations were made during the 1959 and 1960 growing seasons regarding the types, incidence, and importance of red clover diseases in Michigan. The following pathogens were identified: Phoma sp., Ascochyta sp., Kabatiella caulivora (Kirch.) Karak., Erysiphe polygoni DC., Cercospora zebrina Pass., Stemphylium sarcinaeforme (Cav.) Wiltshire, Pseudopeziza trifolii (Biv. - Bern.) Fckl., Uromyces trifolii (Hedw. f.) Lév., Sclerotinia trifoliorum Eriks. Virus and bacterial diseases were also noted. The study showed that the above pathogens were coextensive with red clover culture throughout the State. U. trifolii and the leaf-spot diseases were of moderate to major importance in 1959 and 1960.

Red clover, Trifolium pratense, has maintained an important position in Michigan agriculture for many years as a seed and forage producer. Field observations have indicated that diseases may be responsible for considerable reduction in the general productivity of this plant. To obtain information regarding the types, incidence and importance of red clover diseases in Michigan, observations were made in first crop year red clover fields located throughout the State. Sixty-seven fields were periodically examined during the growing season of 1959, and 34 in 1960, the latter fields being confined to the Lower Peninsula only.

Identification of the various fungus diseases was based on fungus characteristics observed by sectioning leaves and stems showing lesions or fruiting structures. Virus and bacterial diseases were identified by general symptom expression.

Virus Diseases: Red clover plants showing symptoms of virus infections were observed in most fields. Symptoms were common on regrowth after the first cutting (about June 20), and again in late summer when heads first began to dry. At these times 75% of the plants in some fields showed virus symptoms. However, as plant growth increased during mid-summer, the symptoms disappeared. In general, virus diseases appeared most common in the southern half of the Lower Peninsula.

Bacterial Diseases: Evidence of foliar infections by bacteria was uncommon. When symptoms were observed, they were confined to a few scattered plants in a field. The specific bacteria involved were not identified.

Black Stem and Leaf Spot: The Phoma sp. and Ascochyta sp. complex was present throughout the State, but was variable in incidence and severity. Blackened stems were most commonly observed just prior to the first cutting. In a few fields nearly every stem was affected but in other fields stems were lightly infected or showed no symptoms. Foliar symptoms were generally light, and showed the highest incidence on second growth. This disease appears to be of minor to moderate importance in Michigan.

Northern Anthracnose: Kabatiella caulivora (Kirch.) Karak. was found in most fields. Except for a few fields in the Lower Peninsula which had severe infections, the disease apparently caused only minor damage during the 1959 and 1960 growing seasons. Diseased plants were most common during late summer.

Powdery Mildew: Erysiphe polygoni DC. was found coextensive with red clover culture throughout the State. Infections were light to moderate during 1959, but severe in 1960. The degree of injury caused by this fungus is unknown.

Cercospora Leaf Spot: Cercospora zebrina Pass. was commonly observed in all fields examined, and was one of the more destructive foliar diseases in the Lower Peninsula during 1959 and 1960. The characteristic angular lesions often coalesced causing necrosis of many leaves on the plant. Injury was less severe in the Upper Peninsula. Disease symptoms developed most commonly from mid to late summer.

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Stemphylium Leaf Spot: The target-like lesions of Stemphylium sarcinaeforme (Cav.) Wiltshire were observed in fields throughout the State, and were most abundant during July and August on leaves of the second growth. Considered alone, this disease caused a moderate amount of damage.

Common Leaf Spot: Of the leaf spot diseases observed, Pseudopeziza trifolii (Biv. - Bern.) Fckl. was one of the most prevalent. The disease was especially common and injurious during 1959 in the Lower Peninsula. Fields with severe infections showed considerable leaf necrosis, the leaflets often being covered with lesions containing the dark-brown apothecium in their centers. In general, this disease was of moderate to major importance in the southern Lower Peninsula, and of lesser importance in the rest of the State.

Rust: Severe infections of clover rust, Uromyces trifolii (Hedw. f.) Lév., occurred throughout the Upper Peninsula in 1959. The stems, petioles, and leaves of infected plants were densely covered with uredial pustules. This disease was uncommon in the Lower Peninsula during 1959. In 1960, however, the rust was common in the southern Lower Peninsula, a few fields being severely infected. Observations were not made concerning the incidence of this disease in the Upper Peninsula during 1960. In general, clover rust appeared to be of major importance in some areas of Michigan.

Sclerotinia Root Rot: Sclerotinia trifoliorum Eriks. was found in a few fields in the southern Lower Peninsula during the spring of 1960. Damage observed was generally minor, except for a single field in which three to four plants per square yard were wilted and dying from infection. Apparently this disease causes some injury in Michigan, however, more extensive observations would be necessary to determine the extent of such injury.

CONCLUSIONS

On the basis of observations made during 1959 and 1960, the leaf spot diseases, taken collectively, are of major importance to red clover production in Michigan. Leaf necrosis caused by numerous lesions of P. trifolii, S. sarcinaeforme and C. zebrina were common. Frequently these three species of fungi appeared on a single leaflet. U. trifolii was also of major importance during 1959 in the Upper Peninsula. The severe defoliation caused by these pathogens in some fields indicates that diseases are of considerable importance in red clover forage and seed production in Michigan.

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GRAPE MILDEW AS VIEWED IN THE EARLY AGRICULTURAL PRESS OF CALIFORNIA

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That grape powdery mildew probably was not yet recognized in California in 1854 may be inferred from an article on The Grape Disease in The California Farmer of July 27, 1854 (v. 2, p. 28) describing the ravages of the disease in France and Portugal and the sulphur dust treatment recommended for its control. Even as late as 1860 there is no mention of powdery mildew in an article on The Culture of the Vine in The Scientific Monthly (v. 1, p. 68, 1860) of which W. B. Ewer was the alert and enterprising editor. It is stated that, "The grape grown here attains a peculiar perfection, free from the blights which threaten it elsewhere, owing to the dryness of the climate."

In the 1859 Transactions of the California State Agricultural Society (p. 199), Wilson Flint records that in 1859, "...an intelligent vintner of Los Angeles reports..."loss from...a species of mildew,..." and (p. 99) J. S. Silver and F. W. Hatch, members of one of the society's visiting committees, found in Sanford's Shell Mound Nursery at Alameda that, "Grapes, particularly the California variety, and gooseberries, are subject to the mildew..." whereas at Beard's at Mission San Jose, "As yet there has been no mildew observed...on the grapes." Previous visiting committees of 1858 did not report seeing mildew on grapes. J. S. Hittell in The Resources of California (1867, p. 197) said in 1862, "The oidium - the disease which has done such great damage in France - appeared in 1859..."

In 1860 members of the agricultural society's visiting committees (1860Trans. p. 55-79) found powdery mildew in vineyards near Sacramento, Ione, Placerville, Coloma, Auburn, Marysville, Los Angeles and Santa Clara. Wilson Flint visited the A. P. Smith ranch near Sacramento on July 6th and (p. 56) found that "The California Mission Grape is slightly mildewed alongside of foreign sorts, which have escaped" and at the L. W. Hooker ranch, July 27, he found the California grapes badly mildewed and none on the foreign vines. On July 30, the committee consisting of Flint, Wheeler, Nevitt and A. P. Smith visited the Norris ranch near Sacramento (p. 57) and found the California variety mildewed and none on the Catawba variety. On July 31 at Major Barbour's vineyard near Ione (p. 58) they found "...California Grapes badly mildewed, Black Hamburgh and Catawba perfectly healthy..." and on August 10, at C. Couvillaud's (p. 67) near Marysville, the "California Grapes are mildewed, ...no appearance of disease on the foreign sorts..." At W. B. Thornbury's vineyard near Santa Clara, Flint (p. 77) again found the California grapes badly mildewed, but not the Catawba variety. At Los Angeles late in August, he noticed (p. 72) at T. J. White's that "There is a little mildew in shaded places" in the vineyard, and at F. Mellus's (p. 74), "The grapes on an arbor were somewhat mildewed, probably owing to being too much shaded."

Crocker, Fox and Bernhard of another 1860 visiting committee stated (1860 Trans. p. 240) that..."the California variety mildews in localities where many foreign kinds escape..." and that,"...east of the Sacramento and San Joaquin rivers...there is less appearance of mildew on the grapes..." than nearer the ocean. They also thought that "...too much irrigation is given the vine, and...in some instances it is the cause of the mildew." G. Webster, et al., of the 1861 visiting committees (1861 Trans. p. 292) referring to the San Joaquin river said, "Grapes do not prosper well upon the river, the mildew spoiling them."

These early observations indicate that mildew appeared first on the Mission or California variety, originally brought in from Mexico to the Missions and that the more recently imported varieties seemed to escape infection at first.

Judge Daniels in 1860 refers in The California Culturist (v. 3, p. 150) to mildew "...which is without doubt the greatest scourge to which the grape vine can be subjected..." and says (p. 151), "I have tried many remedies; the best I have ever known is to sprinkle the whole of the foliage and branches thoroughly with clear water, -make the whole of the vine thoroughly wet of a still morning or evening..." He adds, "and you may kill it by sprinkling sulphur..."

Signing his name Horticola, a writer in The California Culturist of 1860 (v. 3, p. 272) says, "It can be easily ascertained by the aid of a microscope, that...the Erysiphe Communis, which commits such ravages on our sweet peas, our grape vines, and our peach and apple trees, all belong to the large family of Fungi,...There is no doubt but that the fungi are all

[1]Professor Ralph E. Smith died in 1953, leaving this paper and the following four among his unfinished manuscripts. They have been rearranged and edited by M. W. Gardner. Professor Smith was assisted by Miss Kathleen Bosworth in his search of the journal files.

parasitical in their habits... flourish most... when drawing their sustenance from the essential juices of some unfortunate plant."

The agricultural society Transactions for 1863 (p. 167) contain an Essay on the Culture of the Vine by Dr. John Strentzel in which he says, "The only disease to which our vines are subject is mildew. Of the many preventives which have been tested here, sulphur is by far the most efficacious. Its particles coming in contact with the spores of oidium destroy their vitality..." Three dustings are advised. "Others advise the use of salts of copper to which we emphatically dissent." The 1863 Transactions (p. 173) also contain an Essay on Mildew by Judge Daniels referring to it as "...destroying the crop of grapes, as was the case... in the Valley of Santa Clara, to a partial extent, a few years since." In the Transactions for 1866-67, M. P. Wilder, et al. (p. 360) "recommend most strongly... a renewed and sustained effort to combat mildew with sulphur."

Commenting on the short grape crop, Ewer's journal, now called The Mining and Scientific Press, of September 4, 1869 (v. 19, p. 148) quotes from the newspaper, Alta, "The mildew or oidium has appeared in many vineyards, and has not been checked by the application of sulphur, as it might have been." Under the title, Science for Grape-Growers - The Oidium, the March 13 issue (v. 18, p. 163) quotes from J. L. Russell in the February issue of the American Naturalist that the conidial stage of mildew "...looks like strings of beads made of colorless cells, - in this condition known and described as Oidium,..." and later in autumn are formed, "...shining black capsules, or perithecia,..."

In the same journal, again called The Scientific Press, S. H. Herring, in 1870 (v. 20, p. 287) reports that, "Within a few years several grape crops in Alameda and Santa Clara Counties have suffered seriously from the effects of mildew, in some cases resulting in entire loss to whole vineyards... A timely and proper application will insure the grape against mildew. It is advisable to apply it as soon as the fruit buds are formed - the sulphur to be pulverized and dusted on the vine thoroughly, and again when the blossoms are full... It is better to destroy the sporules of the fungus than to wait until the same is grown on to the fruit. When applied to the fruit the sulphur will destroy the mildew, but it also greatly injures the flavor of the grape by adhering to it... Let every culturist experiment in various ways and note results."

A few weeks later the editor, Ewer, of the Scientific Press (v. 20, p. 327, 1870) points out that the grape growers of France had overcome mildew by sulphuring the vines and still later (v. 21, p. 252, 1870) tells how in the Stockton vineyard and nursery of West Brothers, "The vines and fruit are treated with sulphur thoroughly twice, once as the blossom falls and again when the grape is half grown. The sulphur insures the grape again against mildew,... They use about 100 lbs of sulphur to the acre every season--dusting it on from a small, coarse bag." In 1872, a writer in the Pacific Rural Press (v. 3, p. 338), referring to Dr. Strentzel's Alhambra vineyard, says, "The Doctor has also to sulphur his grape vines three or four times during the season to prevent mildew."

In 1879, Dr. A. W. Saxe of Santa Clara (PRP.[2] 18, p. 66, 1879) stated at a meeting of the California Academy of Sciences, "...That unless three successive applications of the sulphur were made... there was no certainty of success,..." and reported "...entire absence of mildew and perfect grapes" on ten-year-old vines where after pruning he had scraped off the loose bark and with a swab applied "...a solution of bluestone, four pounds to five gallons of water,... to the dormant fruit buds and to the entire body of the vine." He had first used this treatment in the winter of 1868, and from that "...date to the present season I have omitted or neglected to apply the bluestone three seasons—1873, '76, '78; the result has been diseased vines and no grapes,... Last February... "his man"... omitted to apply the solution to one row of 12 vines... At this date, July 1st, all the vines are healthy except this row, which is now exhibiting the curled everted leaf so significant of the disease,..."

Two years later the bluestone wash method was also recommended in the first annual report of the Board of State Viticultural Commissioners (1881, p. 45) by C. A. Wetmore, who says, "Where mildew, or other fungoid disease has appeared, I would, after pruning, scrape the bark and wash the vines with a strong solution of bluestone;..." He also quotes from a grower's letter (p. 46) to the Healdsburg Enterprise, that in a fertilizer test, certain vines received barnyard manure and "...every vine so manured mildewed." Whereas the vines receiving "...a mixture of bones and ashes... are very healthy."

Wickson, the editor of the Pacific Rural Press, published on June 5, 1880, a full page article (v. 19, p. 373) on The Mildew of the Vine with an illustration by Prof. Thos. Taylor, of "growth of grapevine mildew as seen with the microscope." He says, "...The season for the

[2]PRP = Pacific Rural Press.

dreaded mildew is just at hand... the trade in powdered sulphur is now active, and its distribution will soon be general throughout the vineyards... Concerning the method of application, it may be said that both the bellows and the dredge are largely used in this state." He again refers to Dr. Saxe's winter wash with bluestone solution as "...worthy of experiment next winter to see if it cannot be used instead of the method of frequent sulphuring."

The editor had earlier shown his interest in the subject and eagerness for new information for his readers by reviewing (PRP. 14, p. 168, 1877) a recent article by Prof. W. G. Farlow of Harvard, "...too technical to be intelligible to the unskilled reader,..." thinking it might mean the California mildew was different from the Oidium of Europe. This did not prove to be true.

Thus the cause and control of grape powdery mildew was well understood as early as 1870, something which can be said of very few other important plant diseases.

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PEACH LEAF CURL AS VIEWED IN THE EARLY AGRICULTURAL PRESS OF CALIFORNIA

Ralph E. Smith

In the early agricultural literature of California, one plant disease shared with wheat smut the most attention. This is the disease of peach trees called curl, curl leaf, curled leaf, or leaf curl.

Charles Mock of Petaluma states in The California Culturist (v. 3, p. 264, 1860) that during the spring seasons of 1853 to 1855, "The American peaches... did not curl... unlike the California peach, which did usually curl." Replying to an Oregon correspondent's inquiry about the blistering and curling of his peach leaves, the editor (J. F. Morse) of the California Farmer (v. 1, p. 186, 1854) referred to it in June 1854 as "the same disease which has appeared in San Jose and other places...". In the same month he also reported (v. 1, p. 172, 1854) the California peaches at Sacramento and San Jose, unlike the imported eastern trees, "...have been most seriously affected with a blight upon the leaves and a loss of the crop."

Writing to the Pacific Rural Press (v. 21, p. 305, 1881) about Curl Leaf and Drainage, N. Jones of Lafayette said, "...I set out my orchard in about 1853... up to 1862 they were affected badly every year." The trees set along the edge of a creek were the worst affected. Severe leaf curl in Sonoma County in 1857 is recorded by S. W. Shaw (PRP.[1] 21, p. 217, 1881).

In the 1883 report of the California State Board of Horticulture, (p. 57), Dr. H. W. Harkness, the acknowledged "...leader of studies in fungus growth on this coast..." said that this disease appeared in the State almost as soon as peaches were planted, adding, "I observed it first in 1855."

Regarding "This affection, to which the tree is subject during the month of May, and by which it often loses all its first leaves...," T. W. Harris writes to The California Farmer (v. 4, p. 52) in 1855, "The surface of the leaves is swollen into irregular and crisp tumors, often of a reddish and of a spongy texture, formed of thickened and succulent cellular tissue... It is confidently stated that soaping the limbs of the trees early in the spring, or washing them with a solution of sulphur and potash, will prevent them from suffering from the curl. Peach trees on plum stocks seem to be nearly exempt from this affection."

In the Transactions of the California State Agricultural Society for 1858 (p. 121), we read that at the state fair, "Mr. A. H. Myers of Alameda... exhibited some specimens of the Malta peach... which he informs us is not subject to the 'curled leaf,' a disease which is very injurious to the peach trees in some portions of the State." In the 1859 Transactions (p. 99), J. S. Silver and F. W. Hatch of the committee to visit Alameda County report that in Sanford's Shell Mound Nursery the peach is "...extremely liable to the 'curled leaf'" and at A. A. Cohen's, in Alameda (p. 100) "...exposed to the strong winds, peaches do not thrive well... The effect of these winds in the production of the 'curled leaf' appears very evident... The writer of this has repeatedly examined the leaves so affected with a strong glass, but has been unable to discover any evidence of its propagation by insects." John Llewellyn of San Lorenzo (p. 102) told them that, "Crawford's Early is less subject to the curled leaf than most varieties. The same may be said of the President..." In E. L. Beard's orchard at Mission San Jose

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[1] PRP = Pacific Rural Press.

(p. 103), "The peach curls badly...", they found.

According to a note in The California Farmer of March 4, 1859 (v. 11, p. 33), "The past two seasons have each been particularly unfortunate for the Peach crop; the trees having been badly affected with the 'curl leaf'...". A Yuba County correspondent of The California Culturist says in August 1859 (v. 2, p. 79), "No variety of peach seemed exempt... seedling peach trees... were even more subject to the curl... than others... I tried an application of lime water to one, sulphur water to another..." with no success. In 1860, another correspondent (v. 2, p. 543) also reported leaf curl worse on seedling trees.

The 1860 visiting committees of the state agricultural society included Wilson Flint and O. C. Wheeler, whose reports in the Transactions for 1860 indicate considerable interest in plant diseases. In July (p. 56) they found "... the peach is nearly a total failure owing to the curled leaf" on a ranch near Sacramento, on another ranch, "... old peach and nectarine trees... have suffered badly this season with the curled leaf" and on another ranch, leaf curl prevalent except on the variety Crawford's Early. The disease was also found in Yolo County and in August near Ione, Coloma, Auburn, and Marysville.

J. S. Hittell, the author of The Resources of California (p. 189, 1867), writing about peaches in 1862, says, "... nearly all varieties suffer with 'the curl,' which has given so much trouble during the last two years, that many of the orchards have been cut down... The varieties most free from the curl are the Late and Early Crawford, the Late Admiral and the Smock." In the 1861 Transactions of the state agricultural society (p. 322), the West brothers reported the varieties Crawford's Early, Myers Rare-ripe, Smock's Free and Snow free from curl. Years later, W. B. West, writing on Curl-proof Peaches for California in The California Horticulturist and Floral Magazine (v. 10, p. 193, 1880) listed eight varieties as resistant in his experimental orchard of 60 varieties.

Since it is not believed that the leaf curl fungus or any of its hosts is indigenous to California, the common assumption has been that this fungus was introduced into the State on some of the first imported nursery trees. However, Dr. John Strentzel, a reliable observer who came to California in 1850, stated in The California Culturist (v. 2, p. 328, 1860), "Native California seedling trees suffer the most from this disease... Isolated orchards planted entirely with imported trees suffered the least... the disease on these is on the increase." Twenty years later he said, (PRP. 20, p. 82, 1880) "In California, previous to the year 1852, a few peach trees were found about the Missions and around Los Angeles... the trees badly affected with curled leaf... Later, when budded, imported trees began to be plenty, their vigorous growth and luxuriant foliage were characteristics distinguishing them from the so-called Mission peach. A few years later, the curl began to appear also on budded trees." This seems to be evident of the early presence of the disease on seedling trees at the Missions. It also may be significant that other early observers reported in The California Culturist (v. 2, p. 79, p. 264-p. 543, 1860) that seedling or so-called California peaches were more severely diseased than the new varieties.

A striking aspect of the history of peach leaf curl in California is the mystery and confusion which for so many years shrouded popular knowledge of its cause. That it was due to a parasitic fungus, which he called Ascomyces deformans, was announced by Berkeley in England in 1857. Furthermore, the fungus can be readily identified under the microscope. There were several men in California before 1880 who had considerable knowledge of mycology and were familiar with the use of the microscope (Harkness, Anderson, Kinne, Moore).

In The California Culturist in 1858 (v. 1, p. 105) a correspondent asks, "What causes the peach leaf curl?" and expresses his opinion that the disease is "brought about by some peculiar state of the atmosphere at a time when the leaves are in just the proper state to favor its development. The disease itself is purely of a fungus character, and withal, contagious. A diseased leaf will communicate the disease or fungi to its neighboring leaf by contact, and with some smooth-skinned fruits, as the nectarine, to the fruit itself."

To this the editor, W. Wadsworth (v. 1, p. 107, 1858), with 20 years of horticultural experience, replies, "We have made the subject of the curl of the peach leaf a study for years... we have repeatedly tried the experiment by budding from a curl leaf tree to a healthy one, and by displacing portions of the bark of a healthy tree, and supplying its place with bark from a curl leaf tree; but in no instance, or by any treatment possible could the curl leaf be disseminated or conveyed to one free from it. We therefore place the seat of the origin of the disease just where the disease appears, in the leaf itself, and nowhere else... No exposure, no situation, no apparent outward condition of the tree is necessary to induce the existence of the evil. It comes where it will, and when it will... we should look to the effects of climate, or heat and cold, upon the leaf organization... We believe then, that the peach leaf curl is produced by

cold or climatic influences; a disease that...is without a remedy." Wadsworth's familiarity with the graft transmissible yellows disease of peaches in the east is indicated by his statement that "...the yellows as a disease is easily disseminated, and always is, when budding is done from a diseased tree to a healthy one. No so with the curl;...".

In 1872, fourteen years later, when Wadsworth was an editorial contributor to the Pacific Rural Press, we find this same article quoted at length and practically verbatim (PRP. 3, p. 408) and a later editorial, also unsigned, (PRP. 4, p. 1, 1872) to the effect that, "We believe therefore that the peach leaf curl is produced by cold or heat, or certain climatic influences entirely independent of actual frost or the attacks of animaliculae...". This reflects no change from his belief, expressed in The California Culturist in 1860 (v. 2, p. 387) that "...until the true cause is discovered, we are inclined...to attribute it solely to climatic influences."

Writing On the Curl of the Peach Leaf in the 1860 Transactions of the state agricultural society (p. 283), James McClelland thinks leaf curl "...is caused by a sudden change of the atmosphere from heat to cold, while the foliage is in a young and tender state of growth... As the curl is caused by the action of the atmosphere..., I believe it to be a matter of impossibility for man to prevent the ravages of curl to any great extent."

Charles Mock, in his Essay on the Peach Curl in The California Culturist (v. 3, p. 264, 1860), gave some theories about its cause and said, "Our only practical remedy is to plant only the varieties that do not curl, and to bud the curling varieties already planted with the non-curling sorts." However, Dr. Strentzel, writing on Peach Leaf Curl in the same journal (v. 3, p. 44, 1860) shrewdly observes, "...it is assumed that the disease is...caused by the germ of certain fungus taking hold and developing itself on the leaf, sometimes also on the fruit."

In 1861, the state agricultural society (Trans. 1861, p. 65) offered among the premiums for the state fair, a set of Downing's complete works for the "...best statement giving the cause and an effectual remedy for 'curl leaf' in peach trees."

Discussion of the matter in the agricultural press continued and, in the early 1880's, there appeared in the Pacific Rural Press and elsewhere a succession of the most fantastic theories and ideas about the cause of peach leaf curl, and the hopelessness of controlling it. "It is caused by a parasitic fungus" said The California Horticulturist and Floral Magazine in 1877 (v. 7, p. 252), of which E. J. Hooper was editor. But perhaps the concept of a fungus was so hazy that such an identification did not mean much more to the growers than it would have fifty years later to say that a disease was caused by a virus. Many of the ideas expressed about the relation of climatic and soil conditions to the disease resembled some of those promulgated by men of scientific training about so-called physiological diseases of plants fifty years later.

James Shinn of Niles, an intelligent and well-informed fruit grower and nurseryman, writing on Diseases of Peach Trees in the Pacific Rural Press (v. 17, p. 371, 1879), said in regard to leaf curl, "There are no remedies, and the causes which produce it are unknown." The next year, 1880, when leaf curl was extremely abundant, Shinn said in The California Horticulturist and Floral Magazine (v. 10, p. 259), "The most competent observers seem to have agreed that it is produced by atmospheric causes....Are there any remedies? No. All attempts at cure are sheer quackery." In the same journal (v. 10, p. 193, 1880), W. B. West, pioneer nurseryman of Stockton, declared in September, 1880, "Never in the experience of the oldest inhabitant has the curled leaf been so bad; varieties that were considered perfectly free from it have curled badly and now stand almost leafless. The disease has been attributed to many causes...the most rational view, in my opinion, is sudden changes of temperature."

Giving his views on leaf curl in the Pacific Rural Press (v. 19, p. 414, 1880), Robert Parke says, "It is caused by excessive moisture and imperfect drainage,... These conditions promote an excessive flow of the sap upwards, which causes an engorgement of the leaf pores, and a sluggish tendency of the circulation...which is greatly accelerated by the hot sun shining on the tender leaves which causes the sap to become thick and gummy."

M. P. Owen (PRP. 20, p. 194, 1880) says, "The cause of the curl is cold weather" and refers to one variety as being curl-proof. Referring to this disease, Mrs. Jeanne Carr (PRP. 20, p. 155, 1880) says "...a few little cuts with a pen-knife quite through the bark possibly arrested the flow of sap at the right time - the new leaves were not curled at all...". A Vacaville reader (PRP. 20, p. 242, 1880) replies, "I have tried the remedy proposed by Mrs. Carr, i.e., by cutting through the bark, and it is of no use... In my opinion there is just one remedy for curl leaf, and that is to plant trees not subject to it." However, the next year, J. F. Ward of Livingston (PRP. 21, p. 345, 1881) recommended, "...to cure the curl-leaf, split the bark on the tree in four or five places with a sharp knife from the ground up as high as you can reach."

At the meeting of the state horticultural society on July 30, 1880, in which Dr. Strentzel spoke on The Peach Tree and Its Diseases (PRP. 20, p. 88, 1880) remarks about leaf curl were

made in the ensuing discussion by E. W. Hilgard, C. H. Dwinelle, James Shinn, A. T. Hatch and W. W. Smith, leading scientific and practical agriculturists of the State. Hilgard said that "a rich soil and rapid growth favor this disease... since I have manured heavily my trees have curled worse than before." Shinn cited evidence of curl being worse on low land, less on benches. Smith said the varieties Early Susquehanna and Alexander were resistant. The discussion centered around resistant varieties as the only control.

In 1881, another bad year for leaf curl, a paper (PRP. 22, p. 2, 1881) presented before the June 24th meeting of the State horticultural society by Leonard Coates, an educated Englishman, successful nurseryman and experienced orchardist, contains ideas typical of the curious blindness to realities and prevalence of opinions and theories about leaf curl within comparatively recent times. "The curl-leaf... is the worst evil with which we have to contend in the cultivation of the peach. Opinions concerning its cause and remedy are extremely different, not to say conflicting... We believe... the cause to be a too great flow of sap at one time...".

During 1881, growers writing to the Pacific Rural Press suggested (PRP. 21, p. 174, 1881), "... the curl-leaf is produced by something like malarial disease, or the tree is infected by malaria." and (PRP. 21, p. 217, 1881), "In my opinion, the curl leaf is caused by cold, damp winds in spring, when the young leaves are putting out" and (J. Allison), "The sap is rushing up through the wood with great force, a thin and watery fluid. In dry, clear weather the evaporation from the leaves must be considerable. The advent of damp weather stops this evaporation; the leaf-cells are ruptured and the curl ensues as a matter of course." How familiar this would have sounded if it had been said thirty years later about sour sap or gummosis!

In despair, growers said (PRP. 21, p. 174, 1881) "... the cause and cure of the disease never has been and probably never will be found..." and (PRP. 23, p. 373, 1882), "The man who could discover the cause and a remedy for this destructive and perplexing disease would truly be a benefactor to his race."

That the cause of leaf curl "... was determined long ago to be a fungus" was proclaimed by G. C. Swan of San Diego (PRP. 21, p. 273, 1881), who quoted from B. D. Halsted that the leaf curl fungus was Ascomyces deformans, or as Dr. C. L. Anderson of Santa Cruz pointed out (PRP. 23, p. 2, 1882), Taphrina deformans. Swan also quoted T. J. Burrill's paper in the 1878 Transactions of the Illinois State Horticultural Society to the effect that the young leaves were diseased when they "first issued from the buds."

Anticipating by several years the spray control method later adopted, and even the discovery of Bordeaux mixture, is Swan's statement that, "one of my neighbors sprayed his trees last season with a solution of bluestone, which proved effectual."

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POTATO BLIGHT AS VIEWED IN THE EARLY AGRICULTURAL PRESS OF CALIFORNIA

Ralph E. Smith

Knowledge of the catastrophic potato late blight disease in Europe and the eastern States in the 1840's and the efforts made to explain and combat it reached California at an early date, but it was not until after 1870 that the disease caused much concern here. Information about the disease was given in The California Farmer and Journal of Useful Sciences in 1854 in two articles, one on The Disease and Preservation of the Potato (v. 1, p. 36), the other on The Potato Disease in Great Britain (v. 1, p. 59), and in the California State Agricultural Society's (1856 Rep., p. 54) Competition Essay on The Potato and the Diseases to Which it is Liable, by William Thompson.

In The California Farmer (v. 1, p. 10-11, 1854) is a public lecture by Dr. Henry Gibbon delivered in October 1853, in which he said, "I am not aware that the potato rot has made its appearance on this coast." In 1858, the editor, probably W. Wadsworth, of The California Culturist (v. 1, p. 158), speaking of potatoes exhibited at the state fair, refers to "... the disease so destructive to this esculent in the Atlantic States, being wholly unknown in California."

However, certain statements in an article on the potato in the January 12, 1854 issue of The California Farmer (v. 1, p. 13) indicate that the editor, J. F. Morse, feared the disease had already appeared. For example, "There can be no greater calamity befall California than to know that this disease was to continue here." and, "We believe it the duty of the cultivator to take immediate measures for their own relief... every feature of this terrible disease should

be made public...and every experiment that indicates success should also be made known." In the June 8th issue (v. 1, p. 180) he is more positive. "This calamity (for the potatoe rot is a calamity) made its appearance during the last year in California and, were it not for the over-abundant crop, would have resulted in a serious evil." Again, in the July 27th issue (v. 1, p. 28, 1854), he refers to potato growers losing entire crops. Presumably this disease that Morse referred to was not late blight and it is not until the 1870's that there is clear evidence of its presence in California.

Dr. Gibbon, in a lecture in 1868 on microscopic parasites before the California Academy of Sciences and published in The Mining and Scientific Press (v. 17, p. 84, 1868), refers to the potato late blight fungus but not to its presence in the State.

There seems never to have been much mystery in California as to the cause of the disease (as compared, for instance, with peach leaf curl) but its control was as hopeless here as elsewhere. Although it has been called the world's most virulent plant disease, and produced national calamities like that in Ireland, not even de Bary, Farlow, or any other scientist of the time was able to suggest any effective method of controlling potato blight.

During the decade of the 1870's, the blight became prevalent and destructive in the coastal potato-growing districts of California. The Pacific Rural Press published numerous articles about it during this period, using such terms as potato panic (v. 7, p. 376, 1874), potato famine, calamity (v. 7, p. 353, 1874) and total destructiveness. San Francisco, San Mateo, Santa Cruz and Monterey counties were the worst affected.

In 1870, The Scientific Press (W. B. Ewer, editor) reported the following interesting information (v. 20, p. 134, 1870) about potato blight: "It was a favored theory with many two or three years since, that the potato disease was due to the degeneration of the plant, and that if growers would go back to the original wild plant, obtained direct from Peru, healthy varieties would be produced. To test this theory, several prominent growers of this esculent have obtained both seed and tubers from this original source; but the results of the enterprise have not been satisfactory, and the theory is now, we believe, well nigh abandoned." Later, the same journal states (v. 20, p. 342, 1870), "It is also claimed by some experimenters that where the female or seed end of the potato only is planted, there will be no rot and the yield will be much better than when both ends are mingled in the same hill." and it quotes (v. 21, p. 20, 1870) from the Rural World that "...a good circulation of air among the tops and the consequent healthy growth thereof is the best preventive of rot."

In 1872, the Pacific Rural Press, which Ewer had split off from the Scientific Press in 1871, refers (v. 3, p. 137, 1872) to the new Early Rose variety as "...excellent in quality and never affected by the long-prevalent disease." Again in 1873, information is quoted from Moore's Rural (PRP. 5, p. 6, 1873) about a variety trial in which "...scarcely a tuber of the Peerless was affected" by the rot which prevailed in the other varieties.

Unwelcome information began to appear in the Pacific Rural Press in 1874. Thus, in the June 6th issue (v. 7, p. 353, 1874), "Sad news comes to us from what is known as the 'early potato district' of California. This district extends from the toll house on the ocean house road to the Half-Moon bay; embracing a country of twenty miles in length and from four to five miles in width. Not more than a week has elapsed since these fields gave every promise of a good healthy crop of potatoes; but at our present writing whole fields are completely ruined; the blackened, withered vines, looking as though their nourishment were suddenly withdrawn, or had even been subjected to fire... A few of the tubers already show unmistakable indication of rot, in fact both vines and tubers are represented as being in a condition similar to those prevailing in Ireland during the potato famine there... The present blight has already assumed the form and dimensions of a calamity." This is perhaps the first definite record of the disease in the State. The editor states in the June 6th issue (v. 7, p. 376, 1874), "...we have reason to believe that the veritable potato rot is obtaining a foothold in the State."

The next year, another outbreak of potato blight occurred. In the Pacific Rural Press of June 12, 1875 (v. 9, p. 392) we read, "In the potato growing districts of Half Moon bay and the Mission - the same spots that were visited by the blight of last season - this year's crop is probably a hopeless failure... During the past week, Dr. Harkness, with other scientific gentlemen, visited the ground of the Almshouse, where the blight was known to prevail, and examined the crop. At a subsequent meeting of the S. F. Microscopical Society, Dr. H. gave the results of his observations, from which we made the following extracts:

'It has been known for many years that the potato rot proceeds from a fungus...which was named Botrytis by Montagne, who first described it. As it has, however, since been discovered that the pest belongs to a pretty extensive family termed Peronospora, it has received the specific name of Peronospora infestans. Its first appearance this season, as far as report-

ed, was on Wednesday of last week, at the Almshouse farm, where its work was accomplished with a rapidity which seems incredible. On the day previous to its appearance, May 26th, the plants were green, and apparently in luxuriant health. On the day following, the fungus had already accomplished its work. The shrunken leaves were of a dirty brown...while the tubers already exhibited signs of decay.

'As this disease is so violent as to cause the destruction of a crop in a single night, it is useless to discuss methods for a curative treatment. The farmer, who has lost the proceeds of his summer's toil between the hours of his sleeping and awaking, is in no proper mood to hear suggestions as to what might have been done to arrest its progress. After exhausting all our resources for acquiring a correct knowledge of the causes of the disease, we should manifestly give henceforth our undivided attention to searching out means for its prevention. Of the many expedients which have been suggested for the accomplishment of this purpose, none have, as yet, been attended with more than a partial success.'" He suggests treating seed tubers in a "...solution of some material which may prevent the germination of dry spores that may be on the surface, and, afterward, to store them in such places as will keep them as free as possible from contagion." He sent specimens to Dr. Farlow at Harvard.

Referring probably to the same districts, The San Mateo Times of August 28, 1875, is thus quoted by the Pacific Rural Press (v. 10, p. 156, 1875), "The crop on the coast is almost a total failure. Whole fields are devastated by a species of rot and the exhalations of the putrid vegetable are so powerful as to be positively detrimental to health... A few miles from the coast all sign of the infection ceases."

According to the Agricultural Notes column in the Pacific Rural Press, blight also occurred in other districts in 1875. O. N. Caldwell (v. 10, p. 322) reported it from Carpinteria, and the Democrat, a Monterey County newspaper, under date of Oct. 9 (PRP. 10, p. 252) reported "...our potato crop this year will be short, much the larger proportion of the fields so employed having experienced this year the potato 'blight.' This pest, we are told, is growing worse yearly."

On Sept. 30, 1875, a Sonoma County newspaper, the Russian River Flag, reported (PRP. 10, p. 236, 1875), "The disease that has so seriously affected the potatoes in the Bodega country, has extended northward, and we hear complaints of its ravages near Healdsburg... the blight injured chiefly the Bodega (red) potatoes. The Indian premium, the early rose and the pink eyes have almost entirely escaped harm."

According to the Petaluma Argus of September 24, 1875 (PRP. 10, p. 213, 1875), T. J. Ables of Tomales, a careful observer, "...informs us that the reports of the destructiveness of the potato blight have not been over stated... The disease is much worse near the coast than further back."

On July 8, 1876, B. D. H. of Pajaro (PRP. 12, p. 29) reported blight present in many fields. On August 29th, C. N. W. of Watsonville (PRP. 12, p. 170) writes that "...out of the 1200 to 1500 acres of potatoes in the Pajaro valley...there is scarcely an acre which is not affected more or less by the potato rot...not half a crop will be realized... This disease of the potato, as Dr. Anderson informs me, is a fungus, the seeds of which exist in the tubers before planting, and which are developed in the new growth by some unknown conditions of soil or atmosphere, and that the disease may and does exist in the plant in a latent form until these supposed conditions give it an active energy, which results in the destruction of the entire plant and reproduces itself in a future crop by the impregnation with its own virus of the growing potato; or in other words, the seeds of the fungus already existing in the tuber, the favorable conditions concurring, they germinate and send forth their gossamer, thread-like growth more or less rapidly, until the whole plant is reached, appropriated, and its destruction completed."

The Pacific Rural Press of August 26, 1876 (v. 12, p. 141) quotes The Democrat of Monterey County to the effect that, "...the Jersey Blue variety seems almost wholly exempt. Side by side with other varieties, its escape has been almost complete."

On November 17, 1877, E. H. Cheney of Sonoma County reported (PRP. 14, p. 306) that "The potato crop is very light in this section. The blight has nearly ruined some fields and injured all of the red variety more or less." He speaks of success with one variety of seed from the East. In the same issue of the Pacific Rural Press (v. 14, p. 312) E. J. Wickson, the new editor[1], refers to Cheney's success with a change of seed source, and quotes from the London

[1]After December, 1875, E. J. Wickson was the actual editor (PRP. 76, p. 306, 1908), though W. B. Ewer's name remained as Principal Editor until March, 1877. Like Ewer's, Wickson's articles are unsigned, and though other writers may also have contributed unsigned articles, it is assumed that all express Wickson's ideas or at least had his approval.

Gardeners' Chronicle that though "... In England the disease has been studied scientifically and practically for 33 years... The fact is that the cure for the potato disease is as yet an unfound blessing."

Showing his interest and alertness in seeking new information, the editor of the Pacific Rural Press (v. 13, p. 1) writes in January, 1877, "Our potato growers are severely troubled with the blight or rot of the tubers. There is great interest manifested in the cause and cure, but so far as we are informed, there is little being done in the way of experiment to overcome the evil. In order to awaken inquiry in this connection and to draw out our growers in writing what observations they may have made, we are going to tell what an English grower has done and the results which he claims. In a late issue of the London Chemical News we read that J. B. Hannay has reached a conclusion that increasing the temperature of the soil by drawing the sun's rays tends to a stronger growth of the tuber and a freedom from disease... Soot is considered by practical men as a preventive of the disease,... In plots of potatoes planted with and without soot added to the soil... the potatoes grown in a dark soil have a warmer climate, so to speak, than those in a light one. The tubers with no soot were weak and had a great deal of disease among them, whereas those which had the covering were larger and nearly all healthy ...". Later in the year the editor (PRP. 14, p. 200,1877) tells his readers, "...the greatest danger seems to lie in the fact that the mycelium, or thread-like germ tubes of the fungus..." hibernate in the potato itself.

Also in 1877, F. Theirwachter, a subscriber at Watsonville, wrote to the editor (PRP. 13, p. 312, 1877) that he had treated his seed potatoes for control of blight, his idea being that soaking the seed in bluestone solution might control this disease in a manner analogous to the prevention of grain smut by similar treatment. The editor was not optimistic in his reply, citing the negative results of numerous experiments along this line reported by the Royal Agricultural Society of England. He also pointed out the fundamental difference between the fungi involved in these two diseases. "The fungus spores are not dusted upon the surface as with the wheat, but the fungus is developed inside the tuber, thus it is beyond the reach of a surface application." And he adds, "We are glad the experiment is undertaken... because by experiment is the true way to test all ideas..." but, "Do not risk a crop or half a crop by the application of a fancied remedy, the nature of which you do not understand. Try a little patch at first." A later report (PRP. 14, p. 8,1877) in the Watsonville Pajaronian was to the effect that Mr. Theirwachter's treated potatoes were 10 days longer in sprouting "...but are looking stronger and more promising in every way."

On December 14, 1879, E. H. Cheney of Sonoma County, responding to a request from the editor of the Pacific Rural Press (v. 18, p. 403, 1879) that readers send him the results of their "...experience and observations upon the potato and its diseases this season." replied that "The blight is more general this season than ever before and its area is constantly extending. Some fields will not pay to dig; other fields will produce about half a crop, while some fields in sheltered locations are but slightly affected... Last fall...I went some miles to secure some of the Bodega Reds from a field that I had watched and which from all appearances was free from blight, and procured fifty bags. I planted them and every hill blighted, while the new varieties in the same field were free from disease...all varieties sooner or later will be affected in the order of their introduction."

Luther Burbank, in an article on The Burbank Potatoes (PRP. 19, p. 83,1880), referring to 1879, says "Last summer while riding through the Tomales and Bodega potato-growing section, I noticed that the blight first attacked potatoes where the ground was hard, poor, or not well cultivated, while those places which escaped the longest were the small patches in gardens about the houses or near the cattle corral where the land was well manured and often hoed."

In the October 16, 1880 issue of the Pacific Rural Press (v. 20, p. 245), the Petaluma Courier is quoted indicating that blight was bad around Bodega and that Orton Hubbell's newly imported Peruvian seed potatoes produced a healthy crop whereas a strip through the center of his field planted with Bodega reds went down with blight. In the next issue (v. 20, p. 264), the editor calls attention to Hubbell's discovery of the supposed blight resistance of Peruvian potatoes as compared with the Bodega reds, saying, "Mr. Hubbell's experiment at Bodega... may prove of great value to all growers." The next week (v. 20, p. 276), the Petaluma Courier is again quoted, "The potato blight has been more destructive along the coast this season than ever, many fields having been entirely ruined...the blight always occurs during foggy weather, or immediately after a succession of foggy days;...it comes on suddenly, and whole fields are destroyed in a single day."

Wickson's interest in the potato disease is evidenced in the August 7, 1880 issue (PRP. 20, p. 88) by his review of a report on the disease by a Commission appointed by the Royal Agricultural Society of England, pointing out that "it is a sad commentary upon the weakness of

human wisdom that no absolute escape from the evil has yet been made known... how to grow potatoes without having them injured or destroyed is still undemonstrated."

Dr. C. L. Anderson of Santa Cruz closes a long article (PRP. 19, p. 162, 1880) on The Potato - Its Botany, Use and Culture, with the admonition that, "Tubers infected with the 'potato rot', a well-known fungus, should not be used for seed... for it will grow as surely as the potato plant, and if the season is favorable for its growth, will surely blast the whole crop."

E. H. Cheney wrote (PRP. 22, p. 314, 1881) that 1881 was a mild year for blight, and it was late in appearing, but that "Bodega, once so famous for its fine potatoes, has almost ceased to be an important feature in the raising of that valuable esculent. The blight has caused such a shrinkage in the yield and quality of the crop..."

Reflecting his continued interest in the disease, Wickson, the editor of the Pacific Rural Press, reviews in some detail (v. 24, p. 309, 1882) the results of experiments by J. L. Jensen of Sweden who found much less rot in the more deeply buried tubers. The editor was somewhat skeptical, pointing out that little or no rain falls on the crop in California to wash the spores down through the soil onto the tubers.

In the annual report of the State Board of Horticulture for 1883 (p. 52), Dr. Harkness says of this disease, "Little complaint of it has reached us from the interior, but near the coast it has caused much by attacking the leaves and stems and destroying them in a few hours."

Although the discovery of the Bordeaux spray in France in 1885 finally provided an effective control of late blight of potato, Dr. Harkness's statement contains a clue to the way the problem solved itself in California. The large potato production districts, such as Tulelake, Stockton, and Shafter, are now located in the interior and out of the fog belt.

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WHEAT RUST AS VIEWED IN THE EARLY AGRICULTURAL PRESS OF CALIFORNIA

Ralph E. Smith

As in the preceding Spanish era, wheat rust continued to be of great importance in the American era in California. J. S. Hittell, in his book, The Resources of California (p. 176, 1867), states that in 1855, a bad year for wheat, "... both smut and rust raged from Siskiyou to San Diego." The dawning of accurate knowledge regarding cereal rusts and other fungi which was taking place began to reach California in the pages of the agricultural press in the late 1860's, though not without some inaccuracies.

In 1869, an example of the confused ideas of the time concerning wheat rust started in a debate between the writers for two San Francisco newspapers about the nature of wheat rust, which was unusually abundant and destructive that year. The subject was then taken up by the editor, W. B. Ewer, of the Mining and Scientific Press (v. 18, p. 404, 1869) as follows: Rust is, "usually observed when two or more cold, wet days immediately succeed several very warm, bright and dry ones... The plants are chilled while their pores are open. Their vigor is suddenly checked - the plant is sick, and the pestiferous little sporules (which are always flying about and watching for a chance) immediately enter the neglected door and commence their work of destruction." For control of rust he suggested good seed, "to secure a healthy and vigorous growth, such as will resist the insidious attacks,... the sowing of wood ashes over the field", and the use of powdered sulphur, which "has been successfully employed on grapevines and fruit trees to kill the mildew...".

The same journal, now called The Scientific Press, of July 2, 1870 (v. 21, p. 3) contained the following, derived via Silliman's Journal from the Bulletin of the Botanical Society of France: "The effect of barberry bushes in rusting wheat, after having long been accounted a groundless popular superstition, is at length understood and admitted by the Cryptogamists. The botanists used to rebut the charges of the farmers by the statement that the rust in the grain fields and the prevalent fungus of the Barberry belonged to very different genera, and that therefore the one could not give origin to the other. But de Bary in Germany and Oersted in Denmark, following up similar inquiries by Tulasne in France, have concluded that Uredo, Puccinia, and Aecidium are to be regarded not as so many genera, but as three successive forms of fructification of the same fungus, or in some sort an alternation of generations. De Bary ascertained that the spores of the Puccinia graminis do not germinate when sprinkled on the leaves and stalks of the cereal grains, which this rust infests, while they will germinate

on the leaves of the Barberry, and there give rise to the Aecidium berberidis: and the spores of this are equally inert upon the Barberry, but will grow in their turn upon wheat, and there reproduce first the Uredo, or yellow rust, and later the Puccinia graminis, or dark rust." This was rather technical information for an obscure California journal to offer its rural readers more than ninety years ago, and indicates the high intellectual calibre of both editor and readers. A similar article entitled, How the Rust Grows, appeared in 1875 in the Pacific Rural Press (v. 10, p. 211), which had succeeded The Scientific Press in 1871, and of which Ewer was Principal Editor.

In 1870, many farms in the coast country about San Francisco Bay suffered loss of the entire wheat crop from "cryptogamic rust," according to S. H. Herring in The Scientific Press (v. 20, p. 326, 1870), who showed his good sense in the following advice: "If farmers cannot prevent the rust...they can, at least, obviate many difficulties now encountered,...can follow a more diversified and consequently a surer method of farming." Later, he states in The California Agriculturist (v. 4, p. 76, 1874), of which he was editor, "Rust is always worst near the coast, and has seldom been seen in the great hot and dry interior valleys."

At a meeting on June 18, 1872, of the Oakland Farming, Horticultural and Industrial Club (PRP. 3, p. 388, 1872)[1], a long discussion on rust in grain took place. Some of the members brought specimens for examination. Dr. W. P. Gibbons, commenting on the rust fungus, said, "We do not use the microscope enough."

In the spring of 1877 a situation arose which was representative of the status of plant pathology in California at that time. This event illustrated the active public interest which existed here in regard to plant diseases, the alert constructive leadership exercised by E. J. Wickson, the new editor[2] of the Pacific Rural Press, in investigating new agricultural problems, and the presence in the State of a number of men who, though amateurs, were for the time and place versed to an uncommon degree in knowledge of mycology, so far as such knowledge existed anywhere. The situation referred to was the result of an article in the Colusa Sun of March 17, 1877, stating, "Some days ago we heard of a kind of mildew or rust that is attacking all the rankest summer-fallowed wheat. We have heard of it all along the plains from Black's Station, in Yolo county, to a considerable distance above Colusa, and have no doubt but that it extends further up. The stalk near the ground is covered with a slightly reddish-colored coating, which, by scraping off, one can see is injuring the stalk... Our theory of it is that it is a mold, caused by the wheat being constantly wet for the last six weeks." When the editor of the Pacific Rural Press read this he copied the article in his paper and in his words of March 24th (PRP. 13, p. 184, 1877), "We wrote to Mr. Green, editor of the Colusa Sun, asking specimens, which he kindly forwarded by mail."

These specimens and others from other localities were submitted to C. Mason Kinne, Secretary of the San Francisco Microscopical Society, "...an organization which is doing good work for the farmers of our state by the diligent attention which they give to the minute enemies, both of vegetable and animal origin, which prey upon crops and thus frustrate the efforts of the agriculturist." At their next meeting, "...the time was almost wholly engaged in reports and examinations of the specimens of rust and mildew which had been submitted." (PRP. 13, p. 233, 1877). Reports by Kinne and Dr. H. W. Harkness, "...the leader of fungus studies on this coast," were "...accompanied by some excellent figures of the rust and mildew fungi drawn on the wood from the microscope for us by C. Mason Kinne." The figures showed "...the characteristic features and unilocular uredospore of the true 'rust' of the agriculturist, or Trichobasis rubigo-vera of the botanist, one of the phases of Puccinia graminis." Kinne stated that rust infection of the stems caused lodging and quoted Berkeley, "so long as the rust is confined to the leaf, it is, we believe, perfectly harmless." Dr. Harkness stated that, "at least a half-million acres of wheat are in great peril, some of it already destroyed." He identified the mildew as Erysiphe graminis, "which appears for the first time this spring." Subsequent reports were to the effect that the damage to the wheat was caused more by rust than by mildew.

The next year, the editor (Wickson) of the Pacific Rural Press, discussed (PRP. 15, p. 409, 1878), the Rust in Our Grain Fields, illustrating "grain rust as seen with the miscroscope,"

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[1]PRP = Pacific Rural Press.

[2]After December 1875, E. J. Wickson was the actual editor of the Pacific Rural Press, though W. B. Ewer's name remained as Principal Editor until March 1877. Wickson's articles are unsigned and, though other writers may also have contributed unsigned articles, it is assumed that all express Wickson's ideas or at least had his approval.

with part of Kinne's drawings used the year before. Speculating about the mode of infection, he says, "...pseudospores carried by the winds to the leaf, finding favorable climatic conditions, may there vegetate, and the mycelium searching out the stomata no doubt enters and commences its parasitical life... There is no specific for overcoming or preventing the occurrence of rust... Some varieties seem less liable than others, and should be tried persistently..." He quotes B. D. Halsted on the large numbers of spores produced. A few months later, the editor discusses (PRP. 16, p. 337, 1878), Fungus-proof Wheat, an account of work in Europe to obtain varieties of wheat "less subject to baneful influences than the older and more commonly cultivated sorts...one of the principal points being to determine which varieties of wheat are least prone to blight, rust or mildew."

Wickson's qualities of intelligent study, clear thinking, keen observation and acurate common sense conclusions are well shown by his reply, entitled Rusty Ideas about Wheat (PRP. 16, p. 105, 1878), to a subscriber who forwarded to him an Eastern clipping containing fallacious theories about wheat rust. "This is pure conjecture, where it is not nonsense. There is no truth in the use of the simile suggested by the word 'indigestion'... Rust is a parasitic plant. To say that rust results from malnutrition of the plant is a pure assertion. No such thing has ever been demonstrated. In fact, every observer knows that in many cases the rust falls like a scourge upon grain which shows all the marks of being well fed."

In 1879, the Pacific Rural Press again published Kinne's drawings and quoted (v. 18, p. 56.) from a letter from A. Henry, president of the Upper Sacramento Agricultural Society, "Thousands of acres of grain that promised from 30 to 40 bushels per acre are now worthless... The loss to farmers from the effects of rust in this county will be at least 1,000,000 bushels." The spore dust "seems to be odorless and tasteless, yet it affects man and beast strangely... Men who are working in grain badly affected are unable to retain food at times...and it affects the eyes."

In his quest for information on wheat rust for the benefit of his readers, the editor, Wickson, quoted information (PRP. 18, p. 120, 1879),"received by the last Australian mail" from "lectures and discussions on the occurrence of rust by various writers, including Dr. C. Mueke and Mr. McIvor." The importance of the barberry was conceded, "But it has also been shown that the fungus will reach one stage of its growth on other plants than the barberry. It will grow on the wild oats, on weeds and perhaps on many other plants." Rust, said the editor (PRP. 17, p. 264, 1879), "...is a microscopic parasitic plant, growing and reproducing itself upon the substance of the foster plant."

Once more using the drawings made by Kinne, the editor published a full page article in the Pacific Rural Press (v. 21, p. 121, 1881) on Fungoid Diseases of Wheat with correct information on rust, mildew and smut. "There is so much damage done to wheat by the different parasitic fungi which prey upon it, leaf, stem and kernel, that we thought a brief connected account of these pests, with illustrations showing their manner of growth, would be of general value."

The idea of a preventive treatment of some sort for wheat rust had been frequently expressed but never realized. A letter to the Pacific Rural Press (v. 17, p. 242, 1879) stated that in Queensland, treating the seed with carbolic acid had a good effect on rust, as well as being a sure preventive of smut. The editor (PRP. 18, p. 120, 1879) commented as follows: "No specific that would cure rust has yet been announced. Sulphur is the general antidote for fungus growth, and it has therefore been first thought of as a material to save the wheat. But it would seem to be no small task to sulphur fields which contain hundreds and thousands of acres as we have them in this State. An Australian, however,...suggests sulphur fumes would be a remedy for red rust, and thinks that...if sulphur were burned on still nights to windward of rust-affected crops, the smoke would roll over the field in a cloud. He imagines that two or three fumigations would be effectual...". Search for rust resistant varieties and dipping the seed in bluestone were also recommended.

The editor (PRP. 18, p. 257, 1879) relayed further information from Australia that,..."experiments were made by the Rev. E. Cartwright on rust-infected crops by dressing them with salt in solution, the mixture being at the rate of one pound of salt to one gallon of water. The result was completely successful."

The will-o'-the-wisp idea of offering a prize for a method of wheat rust control popped up in Australia. According to the Pacific Rural Press (v. 17, p. 361, 1879), "When the Australians want a thing they believe in paying for it,... Now it is proposed to offer $20,000 for the discovery of a cheap and efficient specific against red rust on wheat."

By 1880, the importance of rust-resistant varieties was more and more appreciated. The Pacific Rural Press (v. 19, p. 81, 1880) stated, "...the escape from rust ravages must come from rust-defying varieties of wheat, rather than from any search for practicable antidotes to

be applied to the field." It was reported (PRP. 19, p. 133, 1880) that Odessa wheat for seed had been received from southern Russia by Mr. A. Gerberding of San Francisco "because of the fame of Odessa as a rust-resisting variety and the fact that the demand for the Anaheim-grown Odessa exceeded the supply." The editor also writes (PRP. 19, p. 152, 1880) about That Indian Rust-proof Wheat, "which has been found to withstand rust so successfully in Queensland" and of which he hoped to receive a sample. W. R. Olden (PRP. 19, p. 186, 1880) stated that Anaheim-Odessa wheat "has been proved by the severest tests to be absolutely rust-proof," and S. Bristol of Ventura said (PRP. 21, p. 4, 1881), "The Odessa wheat...has been grown in Anaheim and in parts of Los Angeles county for six years, and in this county for three years. It has never rusted there nor here." But he warned that the bearded Odessa was susceptible.

For his White Russian wheat seed, A. Gerberding's advertisement (PRP. 21, p. 192, 1881) extolled its large yields, sure crop, "...and above all, its non-rusting qualities. It has been successfully grown in Ventura County, on the sea coast, where, until the introduction of this variety and the 'Odessa,' all other kinds have invariably failed in consequence of Rust."

Charles F. Reed of Yolo County announced (PRP. 21, p. 180, 1881) a Non-Rusting Danish Wheat, saying, "I have shipped a sack by steamer to the Pacific Rural Press," and the publishers, Dewey and Co., offered to send a small sample to any subscriber who would pay the postage, 15 cents.

The Pacific Rural Press (v. 21, p. 113, 1881) carried a front page illustrated article on Leading California Wheats, of which four were the old varieties, White Australian, White Chile, Sonora, and California Club, and the others, "which have won favor for their rust-resisting properties," were Defiance, "Anaheim Odessa, which is widely known from its success on the lower lands of Los Angeles County, where wheat growing has long been thought impossible because of the rust," and "White Russian, which made its first success in this State in Ventura County, and there, by its non-rusting, enabled the farmers to grow wheat instead of barley. The Odessa and White Russian are now being tested in nearly all parts of the State where rust threatens wheat."

Another rust-proof wheat was announced by the Pacific Rural Press (v. 22, p. 68, 1881) resulting from selections made in the bad rust year of 1878 by Mr. S. O. Pugh of Soledad from a field that was ruined by rust. "Among the crop there was a bunch of 16 heads that stood up fresh and green, entirely unaffected by the rust, and when the heads came to maturity, Mr. P. carefully gathered them. The berries from them...were the next season carefully sown by themselves, and so on, until the present season, when he reaps about four tons of grain...". The next year, B. H. Crowell (PRP. 23, p. 470, 1882) described Pugh's Rust-proof Wheat, stating that the original selections in 1878 were from the Australian variety and that, "Mr. Pugh recognized the possibility of this grain being rust-proof and saved the 16 heads... He has this year 60 acres...and until it is proved to the contrary, it can stand as entirely rust proof... Mr. Pugh has reserved the honor of naming it to the Pacific Rural Press." The editor added, "We shall name it in honor of the propagator, 'Pugh's Rust-proof Wheat'."

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WHEAT SMUT AS VIEWED IN THE EARLY AGRICULTURAL PRESS OF CALIFORNIA

Ralph E. Smith

Bunt, or stinking smut of wheat, a fungus disease due to Tilletia species, caused widespread alarm and concern among growers in the 1850's and 1860's, especially in the Sacramento Valley. However, bluestone seed treatment for control of smut was fairly well understood practically from the time of its first appearance in California.

The California Farmer (J. F. Morse, editor) stated in June 1854 (v. 1, p. 194), "There can be no doubt but that there will be more or less Grain lost by smut and rust, chiefly by smut," and in 1855 (v. 4, p. 17), "In relation to grain, very much has been injured by smut, particularly that grown from Australia seed. Some farmers have not made more than half crops. From the Italian seed, Major P. B. Reading...has a very fine crop, without the least smut, whilst acres of the Australian is very much injured."

J. S. Hittell, in his book, The Resources of California (1867, p. 176), refers to 1855 as "...the worst year for wheat we have ever known in California, when both smut and rust raged from Siskiyou to San Diego..." In the Transactions of the California State Agricultural Society for 1866-67 (p. 217), Crane and Bingham record that during the years 1853, 1854, 1855 and

1856, "...our grain was badly injured by smut...". In the 1861 Transactions (p. 331), Major John Bidwell, a reliable observer, said he first saw the disease in 1854 in the Sacramento Valley.

O. C. Wheeler of the visiting committee of the state agricultural society reported in the 1858 Transactions (p. 168), "In...Sutter County...some fields are injured by smut" and (p. 171), referring to a 300-acre field containing three kinds of wheat, "The Australian exhibits some smut, the Club less, and the Sonora none at all... The Committee have found nearly the same result in all parts of the State." On another farm in the same region (p. 172), "The proprietor of this place is also making several interesting experiments with Wheat, with a view to ascertaining the variety which...will yield most and smut least. The Sonora Wheat is entirely free from smut... All other varieties have some smut." Wheeler's committee found one farmer (p. 220) who said "that the soaking of seed-wheat in blue-stone, or salt-petre, or lime, or any other mode of preparation, is no antidote to smut." However, Hutchinson and Green's grain farm (p. 237) in Yolo County had "...fifteen hundred acres in grain,...nine hundred and seventy of wheat... No portion of the wheat is injured by smut. They use vitriol, or blue-stone, in preparing their seed, and believe it a thorough preventive of smut when properly applied." In the same Transactions (p. 346), M. Walthall of Stockton said, "Seed-wheat I prepare in this way:...wash in clear water, if there be smut in it, and soak it in very strong bluestone water - say one pound to eight gallons...about twenty minutes, then roll it in fresh slacked lime...".

In The California Culturist for August, 1858, the editor, W. Wadsworth, has this to say (p. 126) about Sowing Smutty Wheat, "Smut is but a vegetable, a plant, parasite, fungi or mistletoe, just as easily propagated as wheat, and thrives well in soil prepared for wheat, and is ready to attach itself to and destroy the wheat crop... To destroy the vitality of this vegetable fungi before sowing the wheat becomes an important matter with the grain grower." This was remarkably up-to-date information in 1858 and indicates Wadsworth's familiarity with English or European literature. The article goes on, "Countless experiments have been instituted, and we can avail ourselves of the experience of others in this respect,...inasmuch as an almost certain remedy or preventive has been discovered by these experiments. It is simple, not expensive and easily put in practice; has long been in use at the east, as well as in England... It consists in soaking the seed wheat in a solution of blue vitriol in water, and afterward drying, by rolling or dusting it in air-slaked lime or dry ashes before sowing. This is a simple and most effectual remedy, and should be adopted by everyone whose wheat crop is likely to suffer from this infection." Later in the same year (p. 222) he warns, "It is of the utmost importance ...that the seed be entirely exempt from smut."

In the January 1859 issue of The California Culturist (p. 350), Wadsworth repeated the information about the smut fungus "that fastens itself upon the stalk, and also appropriates the entire interior of nearly every grain upon which it takes a hold,..". He described an experiment by T. J. Jones of Contra Costa County in which the seed from a dozen smutty heads of wheat was divided into two lots, one planted in its natural condition, with the result that all the wheat produced was smutted, the other soaked in a solution of blue vitriol and planted alongside the first, the resulting crop being scarcely affected at all with smut. In the December 1859 issue (p. 255), Mr. S. Thompson of Suscol, Napa County, says, "The White Mediterranean wheat...is not liable to smut."

In an address at the State Fair of 1860 reported in the 1860 Transactions of the State Agricultural Society, Major John Bidwell, who came to California in 1841 and was associated with John Sutter in his extensive farming operations in the Sacramento valley, spoke (p. 331) as follows about wheat smut: "...this most valuable of all the cereals is subject to a blight, or disease, called the black ball or smut, which almost threatens its existence. Whole fields are swept away. Many remedies have been proposed, but, on account of our ignorance of the cause, they are partial, not specific - and thus our knowledge on this, as on many important questions relating to agriculture, is altogether empirical. ...Its sudden appearance in California is worthy of remark. From the year eighteen hundred and forty-one to the present time I have been familiar with the cultivation of grain in this country. Its first appearance, at least in the region where I reside, was in eighteen hundred and fifty-four, and it originated in a field sown with Australian seed. But every kind of wheat seems subject to it, unless it be the Turkish Flint... Experience and observation have shown that wheat imported from Chile and Australia escapes the ravages of the smut for one, two, and sometimes three years... Wheat has been subject to this destructive disease in different countries, and various remedies prescribed. More than sixty years ago English farmers were accustomed to wash their seed wheat in lye of wood ashes, lime water, or a solution of arsenic, and, it was thought, with perfect success. And farmers, both of Europe and America, have, from time to time, as one remedy would

seem to fail, substituted others with beneficial results, such as soap-suds, slackened lime, salt water, chamber lye, caustic lime, and blue vitriol. The last named is, beyond question, the most efficacious, (although it has often been known to fail) and should not be discarded until something better has been discovered to take its place... He who shall discover a sure antidote to this apparently unconquerable evil, will deserve to have his name enrolled high on the list of those who have earned the proud title of benefactor of mankind." It is interesting to note that it was during just about this same period that the nature and life history of the wheat smut fungus was being worked out in Europe.

That correct, and fairly complete information about the cause, nature and control of stinking smut of wheat was available in California as early as 1858 has been indicated. That such knowledge was general among grain growers, however, was far from true.

In 1870, W. B. Ewer, editor of The Scientific Press (v. 20, p. 86), emphasized the importance of bluestone seed treatment, discussed (v. 20, p. 39) how to clean smut from wheat to be used for food, and published an article by T. Rowlandson (v. 20, p. 114) on the history of seed treatment with brine and with copper. The brine treatment, he said, was discovered, according to Jethro Tull, as a result of sowing wheat that had been taken out of a ship sunk near Bristol.

A Santa Clara County subscriber to the Pacific Rural Press, a new weekly which Ewer had split off from the Scientific Press in 1871, asserted (PRP.[1] 3, p. 152, 1872), "By actual experience we know that saturating the seed in sulfate of copper will, to a great extent, prevent smut in wheat." In the face of much seemingly overwhelming evidence in favor of bluestone treatment, another subscriber (PRP. 3, p. 210, 1872) makes this statement, "In 1869 I sowed five acres of wheat which had been soaked in bluestone... when the grain was ripe there was smut scattered throughout... The following year, 1870, I sowed the same piece of ground... without using bluestone, and did not find one head of smut...".

In the same issue (PRP. 3, p. 216, 1872), W. Wadsworth, formerly editor of The California Culturist and now an editorial writer for the Pacific Rural Press, attempted to set the matter straight by repeating verbatim and anonymously some of his explanation of the nature of wheat smut he had published 14 years earlier in The California Culturist (v. 1, p. 126, 1858) and had this to add, "...though seed may appear sound, if examined by a microscope, will have a surface more or less affected by smut. Full smut grains will not vegetate or produce wheat; but what we do say, is, that they will produce smut plants or fungi, to seize upon the wheat at the proper season. Hence the necessity of destroying by some application this foul plant, the germs or seeds of which are in countless numbers found on and in the kernels of what is known as smutty wheat, and no application has been found more efficacious than blue vitriol...". Certainly this demonstrates an intelligent interest in the smut problem on the part of the farmers as well as Wadsworth.

In 1874, the Pacific Rural Press[2] contained much discussion of wheat smut, its cause and control. It was pointed out (v. 7, p. 291, 1874), probably by Wadsworth, that, "Smut itself is a plant that attaches itself to and insinuates itself into the kernels of wheat and has its own minute roots independent of the roots of the wheat," and a few pages later (v. 7, p. 337), "It is not supposed that any condition of the atmosphere creates this fungus; but a moist heavy atmosphere undoubtedly favors the growth of the spores which adhere to the grain when sown."

Another article in 1874 was inspired by the following inquiry submitted by a San Jose subscriber (PRP. 7, p. 361, 1874). Both question and answer are indicative of the high degree of intelligence which existed here at this early date regarding such an obscure matter. "I want more light on the subject of smut... Supposition in this matter will not help us much. Farmers want the reason for these things. When the real cause is once known to a certainty, a remedy may soon be obtained." In reply the editor, or more likely, Wadsworth, quoted from Professor Brewer of Connecticut, "who is considered good authority in such matters": The smut fungus "...gains access to the plant through the seed. The spores are sticky and adhere to the sound grain at harvest or thrashing, and are sown with the seed wheat... The spores may be killed and the crop saved by soaking seed wheat in strong brine, or in a weak solution of sul-

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[1] PRP. = Pacific Rural Press.

[2] W. B. Ewer's name appears as Principal Editor of the Pacific Rural Press. He had (v. 1, p. 8, 1871) as regular correspondents: Prof. Ezra S. Carr, W. Wadsworth, T. M. Logan, O. C. Wheeler, E. S. Holden, M. D., J. S. Harbison, S. H. Herring, Dr. Anderson, and Messrs. Hoag and Barnum. After December, 1875, E. J. Wickson was the actual editor (PRP. 76, p. 306, 1908), though Ewer's name remained as Principal Editor until March, 1877. Like Ewer's, Wickson's articles are unsigned, and, though other writers may also have contributed unsigned articles, it is assumed that all express Wickson's ideas or at least had his approval.

phate of copper,...". Using such terms as morphology and metamorphosis, he then endeavors to explain how the grain is converted into a smut ball, resorting to speculation which must have left the grower even more confused.

To T. M. Phelps, of Sycamore Slough, a correspondent of the Colusa Sun, who claimed he found sound grains and smut balls in the same head of wheat and questioned its systemic nature, the editor (Wickson) of the Pacific Rural Press (v. 14, p. 72, 1877) directed this explanation: "When it becomes ripe the fungus bursts out with a mass of dusty powder. This powder, seen with a microscope, is shown to be composed of countless spores (germs), each particle of the mass of dust thus being able to reproduce the smut in full form whenever it falls upon vegetable tissue fitted for its growth...as smutty heads go through the thresher with the healthy ones, it is plain that the chances are that the wheat which is reserved for seed has some of the dusty spores of the fungus clinging to it... But, before sowing, the farmer bluestones his seed. This solution of copper, being fatal to the vitality of the smut spores, simply assures the farmer that he is not sowing live fungus spores with his wheat. Of course this action will not assure him freedom from smut, for there may be live germs waiting in the soil, and germs may be blown upon the plants from adjoining fields."

In an article on Bluestoning Wheat (PRP. 18, p. 354, 1879), C. F. Reed of Yolo County testified that, as a result of the bluestone treatment, "...last season, in a field of 4000 acres, not a single grain of smut has been found." His method, "...in use for many years...", was to dissolve 3 1/2 pounds of bluestone in 6 buckets of water, empty 15 sacks of wheat, or nearly a ton, into a strong tight box, and with the bluestone solution, "...sprinkle the wheat in the box, then with a scoop shovel I have the whole quantity thoroughly mixed so as to absorb the whole amount of bluestone water,... It is then replaced in bags and ready to sow the next day... There are many methods in use by farmers; most of them exceedingly objectionable, particularly that of soaking a sack of wheat in a barrel of bluestone water and letting it hang and drain... The center of the sack oftentimes receives none..." while grain next to the sack "...is thoroughly soaked to such an extent that it will rot in the ground and not germinate."

E. Harley, another Yolo County grower (PRP. 17, p. 322, 1879), was credited with A New Method of Preventing Smut in which, "he poured over his seed wheat water brought to the boiling point, accomplishing the object as effectively as if the most approved methods for the prevention of smut had been used, and that too without apparent injury to any of the germs of the grain."

Interest in the nature and cause of wheat smut and its control by the blue-stone treatment continued to evoke much public discussion in California. A full page article by the editor of the Pacific Rural Press on Fungoid Diseases of Wheat, illustrated with drawings by C. Mason Kinne, appeared early in 1881 (PRP. 21, p. 121). "The smut which works greatest harm to the grain grower is the smut of wheat, Tilletia Caries, the full life history of which is illustrated in Figs. 2 and 3." For information about the fungus, the editor (Wickson) drew "Mainly upon the writings of M. C. Cooke and of Prof. Farlow, the well-known Mycologist of the Bussey Institute... The spores of the smut fungus are sown with the grain...and they can be destroyed...by treatment of the seed. The history of wheat growing is full of receipts for the destruction of smut, and many nauseous compounds have been used. Wheat growers in this State and elsewhere have finally fixed upon blue-stone...as the most effective and most available material... There are ...different ways of applying the blue-stone solution... Whatever method is adopted, the object aimed at is to bring all parts of the grain into contact with the pickle, and to get the seed again dry without steeping so as to cause germination."

Replying to an inquiry from G. W. Lewis of Santa Barbara County, who reported smut worse in a portion of a field planted when too wet, the editor (PRP. 24, p. 8, 1882) again explained the life history of the fungus: "Smut is a fungus; it is a parasite; it has no independent existence of its own... How the spores of the smut fungus gain access to the wheat plant is not known, nor is it fully known what conditions are necessary for its life and growth. The various methods of treating seed wheat to free the coming crop from smut, all rely upon killing the spores of the smut which adhere to the wheat grains. That they are not all killed, or that the spores lie in the ground, or may be blown upon the growing grain from some other source of infection, may account for the fact that dipping the seed does not always save the product...".

Despite all that was known about seed treatment, Dr. R. Schultz of Shasta County in September 1881 (PRP. 22, p. 195) stirred up a controversy with an article, Shall We Bluestone? He denied the value of this treatment citing experiments in Germany and his own experience. "That this drug will destroy the fungus, nobody will dispute, but this does not involve that it will prevent smut 100 days later on the ripening plant." C. A. Anderson of Plumas County replied (PRP. 22, p. 242, 1881) in October, "The writer asks, why so many failures? I am of the opinion

it is not thoroughly applied. Many persons simply sprinkle it on the wheat in bulk, consequently much of the grain fails to receive a sufficient amount to be of any benefit. I have been bluestoning wheat for a number of years, and have never seen smut on wheat I had bluestoned. My plan is to dissolve the bluestone in water in barrels, put the wheat in and let it remain until the grains are thoroughly soaked."

The following rebuttal to Dr. Schultz was copied by the Pacific Rural Press (v. 23, p. 158, 1882) from the Contra Costa Gazette, "...his denial of the efficiency of bluestone as a preventive of smut in wheat...may lead to some distrust of the well-tested remedy for a disease that for a time made it questionable if wheat could be successfully grown in this State, and it was only the sulphate of copper application that determined that question affirmatively. Every farmer in this vicinity, from 1852 to 1856, well remembers how things went from bad to worse in the way of wheat growing...the efficiency of the bluestone solution treatment was shown by the crop of Mr. Thomas Flournoy at Danville, in 1856...the example of Mr. Flournoy's sound crop that season incited all farmers of the neighborhood, though they paid from 30 cents to $1.00 a lb. for the material, to apply bluestone treatment to the seed; in no instance when properly applied was there failure in raising a good sound wheat crop... We may properly mention here that while Mr. Flournoy is entitled to the credit of first demonstrating in this section the efficacy of the bluestone treatment, the credit of introducing its use in the State in 1854, belongs to another citizen of this county, Mr. George P. Loucks, of Pacheco, at that time a produce commission merchant in San Francisco...". He "...procured a quantity of bluestone and induced some German farmers in Alameda county...to give it a trial, the result of which was entirely satisfactory, ...". Newspaper accounts "...led to its more extensive use the following season of 1855 and '56,...".

Refinements in the methods of application of the bluestone solution to obtain better coverage of the seed surfaces were described in 1882 by William Cook of Dixon (PRP. 24, p. 365, 1882) and William Blackwood of Hayward (PRP. 24, p. 388, 1882). Cook's method was to move the seed wheat through a trough with an auger as the bluestone solution poured onto it from a faucet. Blackwood's method was to empty the sacks into a large cask containing the solution, then draw it off, resack the wheat and wait a day or two before planting.

The treatment of seed wheat with bluestone solution for the prevention of smut thus became standardized and universal very early in California and little change in the practice took place for many years.

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INCIDENCE OF CLOVER PHYLLODY IN RELATION TO POTATO CULTURE[1]

R. A. Kilpatrick and K. W. Kreitlow[2]

Abstract

Clover phyllody seemingly is not widely scattered but is localized. A higher incidence of clover phyllody was found in an area devoted to intensive potato culture than elsewhere.

Clover phyllody has been reported from California (3), Maine (5), Oregon (4), and several European countries (1, 2). Abnormal flower development in white clover (Trifolium repens) and other Trifolium spp., characterized by phyllody and virescence, is generally attributed to infection by a strain or strains of the aster yellows virus (3, 4). Raymer and Milbrath (4) reported an association of aster yellows virus in potatoes (Solanum tuberosum) and phyllody in Ladino clover. Increased prevalence of the potato virus disease in Oregon coincided with increased acreage of Ladino clover. Webb and Schultz (5) observed phyllody in Ladino clover growing on Aroostook Farm, near Presque Isle, Maine, in 1954 and suggested that Ladino clover is a possible reservoir for the virus causing purple-top wilt in potatoes. Frazier and Posnette (2), working in Britain, found red and white clover plants with phyllody symptoms near strawberry (Fragaria x ananassa) fields where green petal disease occurred. They suggested that clovers and various weed hosts are possible reservoirs for the virus causing green petal in strawberries.

The aforementioned observations suggest that Trifolium spp., particularly white clover, harbor a strain or strains of aster yellows virus infectious to other species. Consequently, one would expect to find occasional diseased white clover plants displaying phyllody symptoms wherever forage clovers occur. Strangely, this is not the case. Phyllody in clover species in the United States has been rarely observed in pastures, meadows, or roadside stands even though weed species with pronounced symptoms of aster yellows virus infection were growing nearby. When the disease is observed in white clover, other infected clover plants can usually be found in the immediate vicinity. Thus, rather than occurring widely scattered among clover plants, phyllody seems to be localized. This suggests that the virus is being transmitted by leafhoppers to clover species from a nearby host or hosts harboring the virus. The observations herein reported indicate a possible association between potato culture and high incidence of phyllody in Trifolium spp.

PROCEDURE

Webb and Schultz (5) had earlier reported phyllody in Ladino clover growing adjacent to potato fields on Aroostook Farm in Maine. In June 1956, phyllody was commonly observed by the authors in alsike (T. hybridum), red (T. pratense), and white clovers growing in the vicinity of Presque Isle, Maine. An intensive survey was conducted in 1957 and 1958 to determine the prevalence and distribution of the disease in clovers in Maine and New Hampshire. Observations were made with particular reference to the known center of infection near Presque Isle, Maine. Approximately 150 fields containing white, red, and alsike clovers were examined in an area bounded by Portland and Eastport in eastern Maine, north to Presque Isle, Van Buren, and Fort Kent, and south to Bangor and Augusta (Fig. 1). More than 100 fields also were examined in New Hampshire in an area from Durham to Keene in southern New Hampshire, north to Colebrook, and south along the eastern portion of the State to Dover.

Infection was determined by the presence of characteristic phyllody symptoms as described by Halisky, et al. (3). Diseased red clover flower heads were usually stunted and transformed into leaf-like structures (Fig. 2). Flowers of alsike clover were usually proliferated into well-developed leaflets. Occasionally, a secondary flower stalk and phylloid flower head developed

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[1]Cooperative investigations of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture and Departments of Botany and Agronomy, New Hampshire Agricultural Experiment Station, Durham. Published with the approval of the Director of the New Hampshire Agricultural Experiment Station as Scientific Contribution No. 272.

[2]Plant Pathologists, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Durham, New Hampshire, Beltsville, Maryland, respectively.

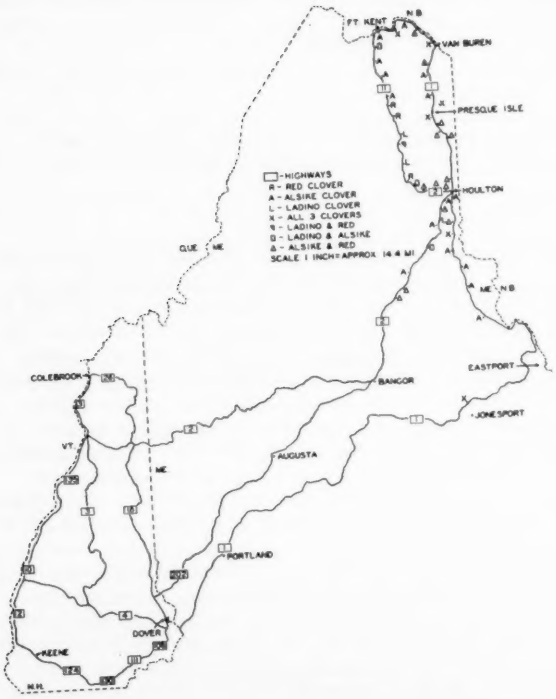

FIGURE 1. Map of Maine and New Hampshire showing known distribution of clover phyllody, 1956-60. (University of New Hampshire Photographic Laboratory)

FIGURE 2. Flowers of red clover showing phyllody, center and right. Note the stunting and leaflet development of flower bracts. Left, healthy flower.

from a diseased flower. Proliferated leaflets in flower heads were first green and turned bronze to yellow. Abnormal flower symptoms were most readily observed in late spring before the first harvest. It was difficult to find abnormal flower heads during August and September.

RESULTS AND CONCLUSIONS

Phyllody was found in the three Trifolium spp. in northern and eastern Maine and in white clover in southern New Hampshire (Fig. 1). Diseased plants were most prevalent within a 50-mile radius of Presque Isle, an area largely devoted to potato culture. Approximately 60% of the clover fields contained phylloid plants. An occasional plant with abnormal flowers was found near Jonesport in the southeastern part of the State. Diseased plants were not found in Maine south of Portland. The disease was not found in New Hampshire until 1960, when phylloid white clover plants were observed in the vicinity of Dover.

Severity of infection varied widely among fields. In 1957, one field of alsike, Ladino and red clover in Maine was estimated to contain 50 to 60% diseased plants. Every flower developing on a diseased plant was abnormal. The same field was examined in 1958 and few clover plants had survived. Halisky, et al. (3) observed that severe phyllody infection eliminated many clonal lines of Ladino clover in an experimental nursery at Davis, California. Diseased alsike and white clover plants transplanted to greenhouses at Beltsville, Maryland and Durham, New Hampshire, were difficult to maintain and usually succumbed to conditions that normally have little effect on healthy plants. This suggests that diseased clover plants in the field may also be short-lived. Since plants with phyllody rarely produce seeds, volunteer reseeding in heavily infected stands is also reduced.

The prevalence and distribution of phyllody in Trifolium spp. suggest that clovers are not commonly infected by strains of aster yellows virus inducing this condition. Except in the situations previously mentioned, phyllody in clover species has been rarely observed in pastures, meadows, or roadside stands. Conversely, high incidence of clover phyllody apparently occurs in some areas devoted to intensive potato culture. The incidence of clover phyllody rapidly diminishes with increasing distance from the potato-growing areas. The evidence suggests that potato culture is in some manner associated with high incidence of phyllody in clovers.

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APHID-TRANSMITTED YELLOWS-TYPE SPINACH VIRUS IN THE EASTERN UNITED STATES

Michael Treshow, A. C. Hill, and W. S. Gardner[1]

Abstract

A disease of spinach causing extensive production losses has been observed in the eastern United States since 1958. Affected plants often appear stunted, and older leaves may develop a yellow to orange, interveinal, chlorotic mottle most pronounced at the leaf tip and margin. The disease was transmitted to spinach by the green peach aphid under controlled conditions. Growth suppression was conspicuous 4 weeks and intercostal chlorosis appeared 5 to 6 weeks after inoculation. Fresh weight of inoculated plants averaged 60% that of the control groups. Results indicate one or more aphid-transmissible, yellows-type viruses are involved.

A leaf yellowing of spinach was observed in 1958, 1959, 1960, and 1961 throughout eastern Pennsylvania and New Jersey. The disease was also observed in Connecticut, New York, and Virginia in 1960. The condition appears to be identical to that reported by Rich (4) to have been prevalent in Connecticut in 1958. Disease symptoms were evident in all spinach varieties observed including Virginia Savoy, Old Dominion, Dixie Market, and Hybrid 7. Many fields were abandoned because the chlorosis made affected leaves unmarketable.

The most conspicuous symptom consists of yellow to orange areas on the older leaves with veins and adjacent tissue generally remaining green. Yellowing first appears toward the leaf tip and margin and eventually may extend over much of the leaf with necrotic lesions frequently developing in the chlorotic areas. These symptoms were present in all the mature plantings inspected in 1960 and were distinct from those of spinach diseases commonly recognized in the area. Studies conducted to evaluate the possible association of this yellowing with a deficiency of certain essential nutrients failed to indicate nutrition as a causal factor. Studies also indicated that fungi were not involved. Investigations were initiated to determine the possible virus nature of the disease.

METHODS

Affected plants were collected from the field and transplanted into pots in a greenhouse. Plants were heavily infested with the green peach aphid, Myzus persicae (Sulzer). Aphids from affected plants were transferred to spinach (Spinacia oleracea var. Old Dominion) and sugar beet (Beta vulgaris) plants. Each plant was separately caged. In the first trial, aphids were allowed to feed 7 days and then killed with TEPP. The above treatment, with a 2-day feeding period, was repeated in trials II, III, IV, and V. In trials II and V, non-infective aphids were placed on plants from trial I which had developed yellowing symptoms and then transferred to healthy spinach and sugar beet plants. Plant diameter, leaf area, and fresh weight were determined when control plants had reached harvest size.

RESULTS

Three to 4 weeks after inoculation, spinach plants on which aphids from field-diseased plants had fed were substantially smaller than control plants. After 6 weeks, interveinal yellow to orange chlorosis and mottling had developed on older leaves of most plants. Symptoms first appeared toward the leaf tip and margin, gradually extending down affected leaves. Plants were noticeably stunted even when chlorosis was relatively mild. The syndrome was indistinguishable from that observed in the field.

In the first trial, 13 of 14 inoculated spinach plants developed symptoms; in all other trials, 100% of the inoculated spinach plants developed symptoms. Data are summarized in Table 1. Plant diameter, leaf area, and fresh weight were consistently reduced when plants were inoculated using aphids from affected plants. Fresh weight of inoculated plants averaged 60% that of the control groups.

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Table 1. Results of inoculation trials.

Trial	Test plant	Feeding time (days)	Number plants inoculated	Number positive transmissions	Plant diameter	Area of four oldest leaves	Fresh weight leaves
					Percent of controls		
I	spinach	7	14	13	79	70	--
	sugar beet	7	8	0	--	--	--
II[a]	spinach	2	7	7	84	81	--
III	spinach	2	6	6	63	34	53
IV	spinach	2	6	6	73	57	64
V[a]	spinach	2	6	6	77	59	62
	sugar beet	4	6	2	--	--	--
	radish	4	6	3			

[a]Spinach plants from trial I used as source of inoculum.

Inoculated sugar beet plants in trial I remained free from leaf symptoms, and no growth suppression was evident. In trial V, a mild and transient leaf mottle developed in older leaves with vein clearing on the younger leaves of three plants. All six inoculated plants were later distinct from controls only by a lighter green leaf color.

DISCUSSION

In this study, the spinach disease causing leaf yellowing and production losses in the eastern States was transmitted by aphids, recovered, and again transmitted. Symptoms reproduced were indistinguishable from those observed in the field.

Symptoms produced on spinach and sugar beet were, in general, similar to those reported for sugar beet yellows in Europe (5) and the United States (1). They also resembled symptoms of radish yellows described by Duffus (2). Duffus (3) reported both radish yellows and sugar beet yellows as occurring on spinach in California.

In preliminary tests conducted with indicator plants, a chlorotic leaf mottle and seed head abortion were produced on shepherd's-purse (Capsella bursa-pastoris) following feeding by viruliferous aphids. Chenopodium amaranticolor and C. capitatum remained normal. Inoculated radish (Raphanus sativus) plants developed a mild chlorosis. These results suggest that radish yellows may be the virus involved, but additional studies will be required to positively establish the virus or viruses responsible for the disease.

Present spinach-growing practices enhance the possibility of virus spread. Spinach is grown throughout most of the year, and new plantings are frequently made in areas of abandoned or overwintered spinach. Insect control is often neglected in abandoned fields near new plantings. Symptoms on other crops and weeds indicate that they may provide additional reservoirs of inoculum.

Stunting of plants, even in the absence of the yellowing symptoms, is particularly important and probably of greater consequence to production than the more conspicuous leaf yellowing. Resistant varieties may have to be sought, or cultural practices developed to reduce the inoculum potential, if spinach is to be grown profitably in the areas where this condition is prevalent.

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EFFECTS OF THREE ANTIBIOTICS ON TREE DISEASES AND FOREST VEGETATION FOLLOWING AERIAL APPLICATION TO WESTERN WHITE PINE STANDS

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Abstract

First-year observations indicated that the antifungal antibiotics cycloheximide (Acti-dione) and Phytoactin, as used, promoted control only on Cronartium ribicola Fischer. However, the semicarbazone derivative of cycloheximide appeared to effect some control of Rhabdocline pseudotsugae Syd. Phytotoxic responses of forest vegetation other than western white pine were minor for all treatments.

A recent report of success in control of the blister rust fungus (Cronartium ribicola Fischer) by application of the antibiotic Acti-dione to western white pine stems[3] raised hope that antibiotics might control other forest tree diseases. Accordingly, when additional applications[4] of antibiotics were designed primarily to evaluate control of the blister rust disease by aerial spraying, supplemental studies were made on other diseases in the sprayed areas. Three antibiotic materials, cycloheximide (Acti-dione), its semicarbazone derivative, and Phytoactin, were applied separately by helicopter to two stands of western white pine (Pinus monticola) that contained Douglas-fir (Pseudotsuga menziesii), lodgepole pine (P. contorta), ponderosa pine (P. ponderosa, western redcedar (Thuja plicata), and grand fir (Abies grandis). The present report describes 1) the effects these three antibiotics had on native fungi that cause other diseases of forest trees, and 2) the phytotoxic reaction of forest vegetation to the applied materials.

STUDY AREAS

Two spray areas with a total of ten 10-acre spray blocks were selected for this study. One is located at Clarkia Peak near Clarkia, Idaho, and the other along the east fork of Potlatch Creek near Bovill, Idaho. The Clarkia Peak area is situated in a natural stand of small pole-sized western white pine, and the Potlatch Creek area is in a 25-year-old western white pine plantation.

MATERIALS AND METHODS

Three 10-acre blocks were sprayed in each area with Phytoactin at concentrations of 100, 200, and 400 ppm in water solution. Cycloheximide semicarbazone was sprayed on three 10-acre blocks at Potlatch Creek with concentrations of 100, 200, and 400 ppm in an emulsion of 20% oil and 80% water. One 10-acre block at Potlatch Creek was sprayed with a concentration of 100 ppm of cycloheximide in an emulsion of 20% oil and 80% water.

Application rate on all spray blocks was 10 gallons of spray solution per acre. Both areas were sprayed during the first week of June 1959.

During the third week of June 1959, study trees were selected in each spray block to represent four major tree diseases, as follows: Christmas tree needle blight (Rhabdocline pseudotsugae Syd.) on Douglas-fir; the western gall rust (Peridermium harknessii Moore) on lodgepole pine and ponderosa pine; and a needle cast disease (Hypodermella arcuata Darker) and a needle blight (Lecanosticta sp.[5]) on western white pine (Table 1). Observations were recorded also on an aphid (Pineus sp.) associated with the needle blight of white pine.

Study trees were marked for identification and location. Recorded data on each tree included species, crown density, crown color, tree vigor, leader growth, and name of the causal

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[3]Moss, Virgil D., Henry J. Viche, and William Klomparens. 1960. Antibiotic treatment of western white pine infested with blister rust. J. Forestry 58: 691-695.

[4]These tests were carried out by the United States Forest Service in cooperation with the Upjohn Company (manufacturers of Acti-dione and its semicarbazone derivative) and with the Pabst Company, (manufacturers of Phytoactin).

[5]Shaw, Charles Gardner, and Charles D. Leaphart. 1960. Two serious foliage diseases of western white pine in the Inland Empire. Plant Disease Reptr. 44: 655-659.

Table 1. Total number of study trees in both areas by treatment and disease.

Treatment (ppm)	Disease		
	Christmas tree needle blight	Western gall rust	Western white pine needle cast disease
Cycloheximide semicarbazone			
100	10	2	4
200	10	13	14
400	10	9	1
Phytoactin			
100	18	18	20
200	20	19	18
400	16	9	14
Cycloheximide (Acti-dione)			
100	10	10	10
Checks	20	14	20
Total	114	94	101

Table 2. Tree tally showing degree of control of Rhabdocline pseudotsugae[a] on Douglas-fir by treatment and by test area.

Treatment and concentration (ppm)	Number of trees by degree of control within each test area					
	Clarkia Peak			Potlatch Creek		
	no control	partial control	complete control[b]	no control	partial control	complete control[b]
Cycloheximide (Acti-dione)						
100	-	-	-	10	0	0
Semicarbazone						
100	-	-	-	7	2	1
200	-	-	-	9	0	1
400	-	-	-	3	6	1
Phytoactin						
100	8	0	0	8	0	2[c]
200	10	0	0	10	0	0
400	8	0	0	8	0	0
Check[d]	10	0	0	10	0	0

[a] The other diseases have been omitted here because all infected trees were in the "no control" category.
[b] Complete control is suggested here, but more testing is required before these results can be accepted as conclusive evidence of control.
[c] This is suspected to result from drift from an adjoining semicarbazone (400 ppm) block.
[d] Trees in this category were not sprayed but were selected near spray plots well beyond the area of drift.

agent of the disease and its infection intensity. Infection intensity recorded for needle diseases was merely a relative measure of the proportion of needles infected in different parts of the crowns of trees. Presence or absence of aphids was recorded for each western white pine sample tree. Records were kept on all species of forest vegetation showing any symptoms of injury that might be considered a phytotoxic reaction to the antibiotics.

A control plot was established for each study area. Sample trees were selected and marked for identification in the same manner used for the treatments.

All study trees were reexamined during the second week of June 1960.

RESULTS

Disease Control: No indication of fungus control was observed on most of the trees selected to represent specific diseases. The only evidence of control appeared on Douglas-fir trees infected with R. pseudotsugae in plots treated with cycloheximide semicarbazone. The extent and degree of control are recorded for this disease by areas and treatments (Table 2). Where control was observed, it ranged from partial (intensities less than the previous season's infection) to complete (no new infection and/or arrestment of the normal disease process).

Eleven trees infected with R. pseudotsugae in plots treated with the semicarbazone derivative showed some degree of control. Seven of these eleven trees were located in the block treated with 400 ppm cycloheximide semicarbazone. Although these first-year observations are preliminary and inconclusive they suggest that the semicarbazone derivative warrants further study as a means of controlling the needle blight of Douglas-fir.

Three trees, one in each cycloheximide semicarbazone spray block, showed no infection in June 1960 on the 1959 needles and no active infection on older needles. The 1957 and 1958 needles had been infected moderately to severely in 1959. They were still on the three trees when checked in 1960, but the infections were inactive. This suggests that the infection was arrested early enough in 1959 to prevent formation of an abscission layer and the consequent casting of the needles.

Two Douglas-fir trees at Potlatch Creek infected with R. pseudotsugae are shown in the "complete control" category for the block treated with Phytoactin at 100 ppm (Table 2). Spray drift from the block treated with 400 ppm cycloheximide semicarbazone was observed in June 1959 to extend for a distance of at least one chain within the 100 ppm Phytoactin-treated block. Because these two trees were located within the area of drift and since Phytoactin showed no control in any other block, the control found on these two trees is suspected to result from cycloheximide semicarbazone.

No evidence of control of P. harknessii, H. arcuata, or Lecanosticta sp. was detected in any spray block. The aphid population on the western white pine study trees apparently was not affected.

Other diseases, Coleosporium asterum (Diet.) Syd. on lodgepole pine, Didymascella thujina (Durand) Maire on western redcedar, Hypodermella concolor (Dearn.) Darker on lodgepole pine, and Phomopsis boycei Hahn on grand fir occurred only sporadically in both control and treated blocks. Because this distribution was so erratic, no data were taken to make comparisons. However, empirical observations showed no evidence of control of these diseases.

Phytotoxic Reactions: Notes taken in 1959 showed that the leaves of huckleberry (Vaccinium sp.) and of boxleaf (Pachistima sp.) suffered minor burning only in the plots sprayed with cycloheximide and its semicarbazone derivative. This burning probably was a phytotoxic reaction to the antibiotics rather than to the oil emulsion, but the effects of oil emulsions alone on these shrubs have not been studied. The fact that burning was not observed when the blocks were checked in June 1960 suggests that the injured leaves of boxleaf had dropped.

No phytotoxic injury from any of the antibiotics was detected on any conifers except western white pine. A very striking reaction in white pine was noted on all blocks sprayed with cycloheximide and cycloheximide semicarbazone. This injury was produced by the antibiotics rather than the oil emulsion[6]. The injury was equally severe on all blocks regardless of concentration of the antibiotic. One-year and older needles turned yellow several weeks after application and were cast during the first week of July 1959, one month after spraying. However, the current season's needles were not affected by the antibiotics. Whether they are naturally resistant or were still protected by the bud scales at the time of spraying is unknown. On examination in 1960, all affected trees appeared to be healthy and recovering from the foliage loss caused by the antibiotics.

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[6]Conclusions based on unpublished studies by V. D. Moss, United States Forest Service, Spokane, Washington.

THE STEMPHYLIUM LEAF SPOT COMPLEX ON BLUE LUPINE[1]

Homer D. Wells, Ian Forbes, Jr., and John R. Edwardson[2]

Abstract

Strains of Stemphylium botryosum Wallr. highly pathogenic to commercial varieties of blue lupine were identified for the first time in 1960 and 1961 in south Georgia and north Florida. These virulent strains of S. botryosum caused symptoms identical with those of gray leaf spot caused by S. solani Weber. Greenhouse and field studies showed that breeding lines that had previously been proved resistant to S. solani were also resistant to the virulent strains of S. botryosum. It is proposed that the name gray leaf spot be redefined to include the identical symptoms produced by both S. solani and virulent strains of S. botryosum.

INTRODUCTION

When breeding lines of blue lupine (Lupinus angustifolius) were being examined at the University of Florida Agricultural Experiment Station at Gainesville in May 1960, severe defoliation was noted and the few remaining leaves at the tops of the plants showed typical symptoms of gray leaf spot caused by Stemphylium solani Weber (3). A number of leaf specimens were collected, and lesions were surfaced-sterilized and plated on 20% V-8 juice agar. S. solani, the causal organism of gray leaf spot, was not obtained. All lesions, however, yielded pure cultures of a Stemphylium characteristic of S. botryosum Wallr. Subsequently a number of these cultures produced the perfect stage Pleospora herbarum (Pers. ex Fr.) Rabenh.

Previous investigations (3) had shown S. botryosum to be the cause of a disease which was given the name little leaf spot of blue lupine. Past experience had indicated that S. botryosum was not of major importance except where blue lupines were grown adjacent to alfalfa (3). However, there were no alfalfa plantings in the immediate vicinity of the breeding nursery at Gainesville. Apparently the S. botryosum strains involved in the damage were more virulent on blue lupine than those previously observed.

To determine the relative importance of S. botryosum and S. solani in the apparent leaf spot complex of blue lupine, a survey was made during the spring of 1961 in south Georgia and north Florida.

Considerable breeding work has been done on incorporating resistance to gray leaf spot caused by S. solani (1, 2) in a blue lupine forage line (60-206). Since it appeared that S. botryosum might be as destructive as S. solani in the Stemphylium leaf spot complex, experiments were conducted to determine their comparative pathogenicity on a commercial variety known to be susceptible to S. solani and on breeding lines known to be resistant. These tests were the first steps in a search for resistance to the more virulent strains of S. botryosum.

STEMPHYLIUM LEAF SPOT SURVEY IN 1961

On March 23, 1961 observations were made on Stemphylium leaf spot reactions of blue lupine entries in the nursery at the University of Florida Agricultural Experiment Station, Gainesville, Florida. All entries except S-13 and 60-206 were severely defoliated. The 60-206 line is an F_4 progeny of the three-way hybrid (54-202-1-C x Blanco) X (S-13) and has been proved to carry the recessive gene pair gl gl (originally found in S-13) for resistance to gray leaf spot caused by S. solani (1, 2). On March 23, 1961 a common bitter blue lupine planting was observed at Jennings, Florida in which Stemphylium leaf spot was causing some damage. A Stemphylium leaf spot disease survey was made in areas north and south of Gainesville on April 1, 2, and 3. Stemphylium leaf spot was causing severe defoliation in the immediate vicinity of Gainesville, but was of minor importance 40 miles south of Gainesville near Ocala. The Ocala area

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[1]Cooperative investigations at Tifton, Georgia and Gainesville, Florida of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture, the University of Georgia College of Agriculture Experiment Stations, Coastal Plain Experiment Station, and the Florida Agricultural Experiment Station, Gainesville. Published with the approval of the Director of the Coastal Plain Experiment Station as Journal Series Paper No. 85.

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Table 1. Occurrence of Stemphylium solani and S. botryosum in lupine fields in different areas as indicated by recovery from isolations on 20% V-8 juice agar from surface-sterilized lesions typical of gray leaf spot.

Lupine variety	Location	Stemphylium species recovered[a]	
		S. botryosum	S. solani
Breeding lines	Gainesville, Florida	+	+
Common bitter	Jennings, Florida	-	+
Common bitter	Madison, Florida	+	-
Borre	Madison, Florida	+	+
Common bitter	Ellisville, Florida	+	+
Borre	Ellisville, Florida	+	-
Common bitter	High Springs, Florida	+	+
Borre	Archer, Florida	-	+
Borre	Newberry, Florida	+	+
Borre	Newberry, Florida	-	+
Borre	Newberry, Florida	+	+
Common bitter	Raleigh, Florida	+	+
Common bitter	Ocala, Florida	+	+
Blanco	Tifton, Georgia	+	+

[a] - indicates absence of the species and + presence.

Table 2. Variability in pathogenicity of 21 isolates of S. botryosum from blue lupine on Blanco blue lupine.

Reaction classes[a]	Number of isolates in different reaction classes
0	1
0.1 - 1.0	0
1.1 - 2.0	7
2.1 - 3.0	2
3.1 - 4.0	1
4.1 - 5.0	10

[a] 0 = no infection.
5 = severe infection resulting in complete killing.

Table 3. Reactions of seedlings of S. solani resistant blue lupine lines and Blanco blue lupine to six isolates of S. botryosum and one isolate of S. solani 7 days after inoculation.

Blue lupine lines	Reactions[a] to indicated lines of S. botryosum						Reactions to S. solani
	60-23-A	60-23-D	60-23-F	60-41-2	60-41-3	60-41-8	
S-1	-	-	-	-	-	-	-
S-4	-	-	-	-	-	-	-
S-5	-	-	-	-	-	-	-
S-6	-	-	-	-	-	-	-
S-7	-	-	-	-	-	-	-
S-8	-	-	-	-	-	-	-
S-9	-	-	-	-	-	-	-
S-10	-	-	-	-	-	-	-
S-13	-	-	-	-	-	-	-
WH-1	-	-	-	-	-	-	-
Blanco	+	+	+	+	+	+	+

[a] - = no infection.
+ = severe damage resulting in defoliation and death of seedlings.

was undergoing a severe local drought, which might account for the lack of Stemphylium leaf spot. Disease severity steadily decreased with distance north of Gainesville. Since blue lupines north of Gainesville were also later in maturing, the disease could have built up to damaging

proportions after this date. This was the case in a Blanco blue lupine seed field at Lakeland, Georgia, which contained only a moderate amount of Stemphylium leaf spot in early April, but was almost completely defoliated by the disease when revisited on May 2. Thirty isolations per field were made from "gray-leaf-spot-like" lesions from specimens taken from 14 of the fields visited during the spring of 1961. The lesions were surface-sterilized in an 0.8% sodium hypochlorite solution and plated on 20% V-8 juice agar. The small sample is not adequate for statistical consideration, but the data (Table 1) do indicate that S. botryosum and S. solani are both important as causal agents of the Stemphylium leaf spot complex in blue lupines.

PATHOGENICITY

To determine virulence of strains of S. botryosum on blue lupines, two experiments were conducted in which 21 isolates of S. botryosum from gray-leaf-spot-like lesions on blue lupine were used as inoculum on Blanco blue lupine seedlings. The inoculation technique described by Forbes, et al. (2) was used in all pathogenicity tests reported. Disease reactions were determined 5 days after inoculation. Results of these tests (Table 2) demonstrated a wide variation in pathogenicity of S. botryosum as shown by the fact that one isolate failed to infect the test plant whereas ten proved to be capable of killing Blanco blue lupine seedlings.

Since highly pathogenic isolates of S. botryosum were shown to be present on blue lupine, a search was initiated within blue lupine breeding lines at Tifton to locate resistance to pathogenic strains. Therefore, six of the highly pathogenic isolates of S. botryosum were compared with a known highly virulent isolate of S. solani on ten blue lupine breeding lines known to be resistant to gray leaf spot caused by S. solani. Reactions resulting from these inoculations (Table 3) proved that all lines resistant to S. solani were also resistant to S. botryosum, whereas Blanco blue lupine was highly susceptible to all isolates of S. botryosum and the standard isolate of S. solani. The mode of inheritance of resistance to gray leaf spot has been determined for only S-13. This resistance has been shown to be conditioned by a single recessive gene pair gl gl (2). An additional study in which Blanco and the S-13 line were inoculated with ten isolates each of S. solani and S. botryosum from "gray-leaf-spot-like" symptoms of blue lupine was made. In all instances the S-13 line was highly resistant to all isolates of both species, whereas Blanco was highly susceptible to all isolates.

DISCUSSION

Strains of S. botryosum highly pathogenic to commercial varieties of blue lupine were identified for the first time in 1960 and 1961 in south Georgia and north Florida. Field observations and greenhouse tests demonstrated that S. botryosum is as destructive as S. solani on commercial blue lupine varieties. The fact that an advanced forage breeding line (60-206) which had been developed for resistance to gray leaf spot, caused by S. solani, and anthracnose, caused by Glomerella cingulata (Ston.) Spauld. & Schrenk, was also highly resistant in the field to virulent strains of S. botryosum is extremely fortunate. The fact that numerous unrelated breeding lines resistant to S. solani were also resistant to virulent strains of S. botryosum leads one to suspect that the genes which confer resistance to S. solani in these breeding lines also confer resistance to virulent strains of S. botryosum. However, genetic tests will be necessary to support the suspicion of "pleiotropy" and to eliminate other possible explanations.

Since the more virulent strains of S. botryosum cause symptoms that cannot be distinguished from gray leaf spot caused by S. solani, it is proposed that the name gray leaf spot be redefined to include the identical symptoms produced by both S. solani and virulent strains of S. botryosum.

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MYROTHECIUM RIND ROT OF CANTALOUP

D. M. McLean and Bailey Sleeth[1]

Rind-rotting lesions are sometimes serious on certain varieties of cantaloups (Cucumis melo var. reticulatus) in the Lower Rio Grande Valley of Texas. Infected fruits are discarded in the field, but incipient infections are difficult to detect and shipping losses result. Scattered greenish-black, viscid, loosely woolly, white-fringed bodies (sporodochia) 1 to 1.5 mm in size sometimes develop superficially on such sunken fruit lesions. Sporodochia may coalesce into a charcoal-black viscid sheath (Fig. 1C) or dry to a hard flaky mass. Myrothecium was readily and consistently isolated from these spore masses. Inoculations from pure culture isolates to unblemished, surface disinfected melons caused a slow rind rot with a shallow to deep, light-colored, rather firm core (Fig. 1D) extending into the flesh. Watery breakdown of flesh tissues was not rapid and off-flavor of infected fruits was not pronounced. Typical sporodochia developed on inoculated lesions from which Myrothecium was readily reisolated. The fungus also has been observed on lesions of honeydew melons. In the fall of 1958, during periods of high temperatures and excess rainfall, infections by Myrothecium of leaves on cantaloup were common. Except for the presence of sporodochia, leaf lesions (Fig. 1A) resemble somewhat the concentric zoned lesions caused by Alternaria. Stem lesions near the crown also have been observed bearing characteristic sporodochia (Fig. 1B).

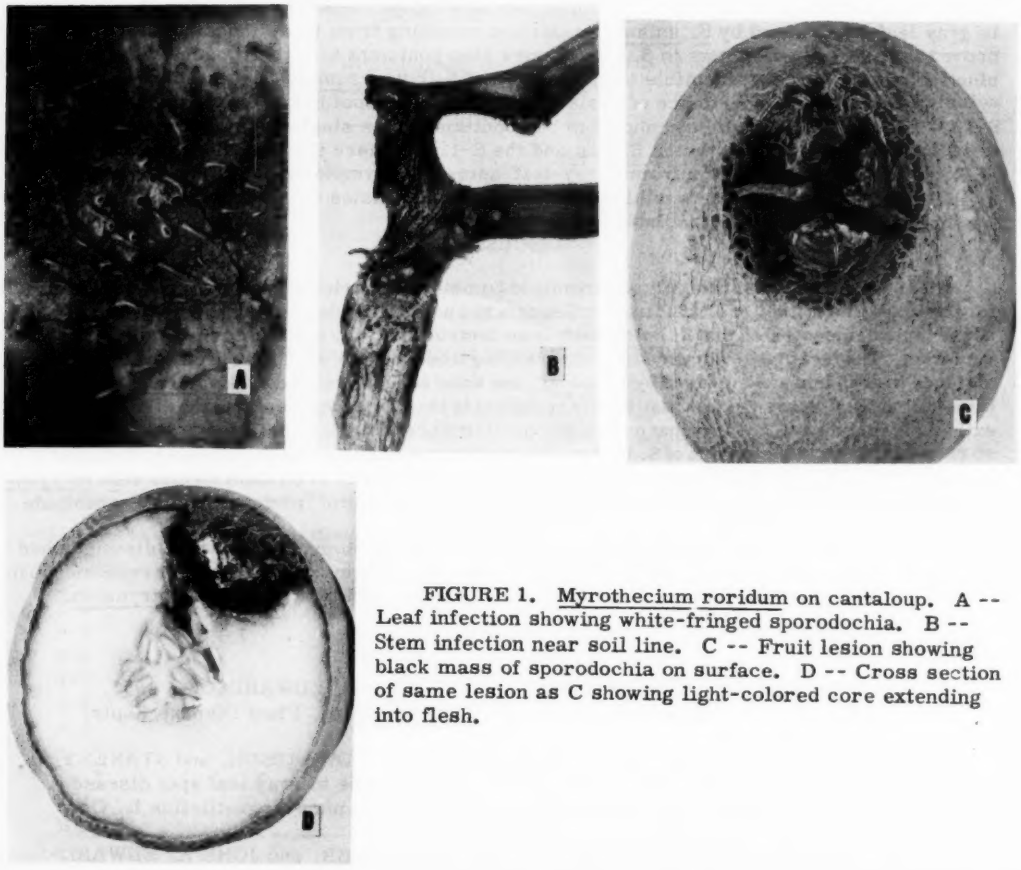

FIGURE 1. Myrothecium roridum on cantaloup. A -- Leaf infection showing white-fringed sporodochia. B -- Stem infection near soil line. C -- Fruit lesion showing black mass of sporodochia on surface. D -- Cross section of same lesion as C showing light-colored core extending into flesh.

[1]Pathologists, Crops Research Division, Agricultural Research Service, United States Department of Agriculture and Texas Agricultural Experiment Station, Substation 15, Weslaco, Texas, respectively.

Myrothecium usually is regarded as a weakly parasitic fungus (1, 3). It is found most commonly on dead or decaying plant parts, but rarely on foliage, and is reported mostly from tropical or subtropical regions. Preston (1, 2, 3) listed a number of exsiccata specimens on diversified hosts. In the United States the fungus was reported only from Texas as a cause of stem canker on tomato (6), crown rotting of greenhouse snapdragons (5), and crown necrosis of Bells of Ireland (Molucella laevis) (4). The fungus has been isolated from tomato fruit and leaf lesions in the Lower Rio Grande Valley of Texas (unpublished data). Apparently Myrothecium has not been reported on Cucumis melo. A specimen of Myrothecium sp. on cantaloup from Mexico intercepted in Texas in 1950, however, is on file in the National Fungus Collections (C. R. Benjamin).

Shape and size of conidia and lack of setae in the white-rimmed sporodochia fitted the description of Myrothecium roridum Tode suggested by Preston (1, 3). Conidia were cylindrical with rounded ends, hyaline at first, becoming pale green to black in mass, always less than five times as long as wide, averaging 5.5-9.5 x 2-2.5 microns. Sporodochia 1 to 1.5 mm often confluent becoming jet black, viscid, globular or conical at first, finally dry, dull black, flattened or slightly concave, with a white margin of flocculent contorted hyphae lacking setae (Fig. 1A). Optimum temperature for growth on potato-dextrose agar, slightly alkaline, was approximately 25° C.

On the basis of these observations M. roridum appears to be commonly associated with rind rot of cantaloups in the Lower Rio Grande Valley of Texas. The importance of the fungus as a primary pathogen is not known.

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STEWART STRAIN OF BARTLETT PEAR SHOWS FIRE BLIGHT RESISTANCE[1]

Roderick Sprague, Archie Van Doren, and Peggybeth Figaro[2]

In 1959 Howard M. Stewart of Wenatchee, Washington brought to the Station scions from a healthy branch growing on an otherwise fire blight-infested Bartlett pear tree. A series of inoculation trials on blossoms resulted in short-lived lesions of blight (Erwinia amylovora) on both the Stewart stock and on standard Bartlett trees in the greenhouse. Nevertheless, these tests during 1960 were suggestive of some blight tolerance in the Stewart strain. Since the pathogenicity of the isolates of blight was not satisfactory, this spring a virulent strain was obtained from P. A. Ark, which has been maintained on young green pear fruits. Blooms in the field were sprayed with sterile distilled water suspensions of blight inoculum from oozing fruits on April 27, May 15 (second bloom), May 18 and May 26. Rapidly moving infections were obtained from the April inoculation on standard Bartletts and, in somewhat reduced extent, on the Stewart strain. Most of the lesions on the Stewart strain died in the fruit pedicel. One invaded the fruit and eventually into the wood, dying out about 1 foot from the place of infection.

Since the April 27 inoculations had been on young Stewart strain pears and on large standard pears, the inoculations on May 18 were applied on a heavily dehorned Stewart topworked tree and compared with inoculations on a less vigorously growing mature standard pear. Even under these unfavorable conditions the Stewart strain developed considerably less blight than the standard.

Table 1 gives a summary of blight losses in several dozen inoculations during 1961.

Table 1. Amount of blight injury in artificially inoculated Stewart strain Bartletts compared with standard Bartletts at the Stewart Ranch, 1961.

Date inoculated	% loss[a] in: Stewart strain	Standard strain
April 27	28.8	78.1
May 15	10.0	26.0
May 18	8.8	17.3
May 26	13.3	21.1

[a] Calculated as follows: Infections that died in the pedicel, 10% loss; lesions causing oozing fruit, 20% loss; lesions that penetrated into the wood beyond the spur but ceased to spread, 50% loss; lesions in wood still active, 100% loss.

Also, young pear fruits were puncture-inoculated from standard and Stewart strain trees in the laboratory with E. amylovora. After 72 hours, lesions on standard pear fruits averaged 18 mm diameter while those on the Stewart strain averaged 14 mm.

In these studies all evidence indicates that the Stewart sport has some tolerance of blight, but it is by no means immune. The fruit seems to be nearly as susceptible as the standard strain but the wood appears to resist invasion by the organism. Several years of observation and trials, including careful study of the growth habits and horticultural merits of the strain, lead to the conclusion that the Stewart strain should be increased for wider trials.

The Stewart strain has blight tolerance that classes it moderately susceptible in the severe trials reported here. However, there appears to be no reason why this strain should not be considered as a replacement for the completely susceptible Bartletts.

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[1] Scientific Paper No. 2125. Washington Agricultural Experiment Stations, Pullman. Project No. 1164.

[2] Respectively, Plant Pathologist, Superintendent, and Senior Technician. Washington State University Tree Fruit Experiment Station, Wenatchee. Sprague and Van Doren assume equal responsibility in this paper, the arrangement of their names being alphabetical.

DISTRIBUTION AND CONTROL OF CERCOSPORA THUJINA ON ARIZONA CYPRESS IN GEORGIA[1]

G. E. Thompson and B. R. Murray[2]

Summary

A needle blight of Arizona cypress was found to be widespread in cultivated plantations in Georgia. Surveys indicated that Cercospora thujina was causing most of the needle blight in all plantations visited. Fungicidal sprays of Tribasic Copper Sulfate, maneb, and Dyrene gave control of the disease in the field and greatly reduced the number of cull trees.

A survey was made in 1958-59 of 85 Arizona cypress (Cupressus arizonica) plantations throughout the State of Georgia, and foliage blight was observed in every plantation visited. Some stands had very light infection while in others infection was severe. The infection varied from 2 to 80% with the majority of the plantations having about 30 to 40% of the trees affected. No plantation was found to be free of this disease, and one stand observed had a 60% loss in a single year. In this case the disease became severe during the unusually wet summer of 1959 when the plantation was 3 years old.

An inspection was made of only a portion of the plantations visited, but all that were checked had Cercospora thujina Plakidas fruiting bodies present on the infected tissues, and infection in all stands visited seemed to be of the same type. Isolated cases of Phomopsis juniperovora Hahn were found, but its damage and distribution was insignificant compared with that of C. thujina.

Re-checks of several Arizona cypress plantations widely distributed throughout the State were made in June 1960, and Cercospora thujina again was found to be the primary causal agent of foliage blight.

An experimental planting of Arizona cypress was established in January 1960 close to a stand infected with C. thujina. An inventory of the planting made on June 10, 1960 revealed that it had a 34% infection by C. thujina.

The future of Arizona cypress Christmas tree production in Georgia depends on finding a control of this disease.

SYMPTOMS

The infected trees exhibit a reddish-brown dying back from the tips of the branches. Infection on individual needles spreads down the needle into the tissues of the twig causing a reddish-brown discoloration of the infected area (Fig. 1A). The twig infections encircle the stem and are reddish-brown at first but older dead twigs become ashen-grey in color. Severely infected young trees may be killed by the fungus.

Conidia are sparse on recently killed needles but become more abundant on older dead needles. Sporulation is more abundant on infected trees in low areas, particularly those near ponds.

THE FUNGUS

The description of the fungus and growth characteristics on artificial media agree with those of Plakidas (1) and Wilson (2). Sections of diseased host tissue, conidiophores and conidia are shown in Figure 1.

CONTROL

Procedure: A plantation of young Arizona cypress trees that had been transplanted the previous January was used in the experiment. The plot was divided into 10 replications of 10

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[1]Cooperative investigations of the University of Georgia College Experiment Station and Agricultural Extension Service.

[2]Professor of Plant Pathology (deceased) College Experiment Station, Athens and Extension Forester, Agricultural Extension Service, Tifton, respectively.

Table 1. Effects of fungicides on Cercospora blight of Arizona cypress, August 1960.

Treatment	Replications[a] 1	2	3	4	5	6	7	8	9	10	Totals	Means
Untreated control	20	28	32	24	25	22	21	26	27	25	250	25.00
Tribasic Copper Sulfate	17	19	18	16	20	16	15	19	14	15	169	16.90
Maneb (Manzate)	16	13	15	14	14	14	13	17	15	16	147	14.70
Dyrene	11	14	13	14	16	18	19	16	16	18	155	15.50
LSD 5%												4.28
LSD 1%												5.79

[a]The data represent a total disease index for 10 trees in each treatment.

Table 2. Disease ratings of Arizona cypress trees sprayed and unsprayed, August 1960[a].

Treatment	Replications 1	2	3	4	5	6	7	8	9	10	% culls[b]
Control	1	4	4	3	1	1	2	1	4	1	52
	2	3	4	1	3	1	2	2	4	4	
	1	3	4	2	2	2	1	1	1	3	
	1	1	1	1	3	4	1	2	4	1	
	4	1	3	4	3	1	3	4	2	2	
	3	2	3	1	3	3	1	4	2	4	
	1	4	3	1	4	3	2	3	3	4	
	1	4	4	4	2	3	3	3	3	2	
	4	3	2	4	3	2	3	2	2	1	
	2	3	4	3	1	2	3	4	2	3	
Tribasic Copper Sulfate	2	1	2	1	2	1	2	2	1	1	6
	2	2	2	1	1	1	2	2	2	1	
	2	2	1	1	3	2	2	1	1	2	
	2	2	1	2	3	1	2	2	1	2	
	2	2	2	2	1	3	1	1	1	2	
	2	2	2	2	2	2	1	3	1	1	
	2	3	2	1	2	3	2	2	2	2	
	1	2	2	2	2	1	1	2	2	2	
	1	2	2	2	2	1	1	2	2	1	
	1	1	2	2	2	1	1	2	1	1	
Maneb	1	2	1	2	1	2	1	2	2	1	2
	2	1	1	1	1	2	2	2	2	3	
	2	1	1	1	1	1	2	1	1	2	
	1	1	2	1	2	1	1	1	2	1	
	1	1	2	2	1	1	1	3	1	1	
	2	2	1	1	2	1	1	1	2	2	
	2	1	2	1	1	1	2	1	1	2	
	2	1	1	2	2	2	1	2	1	2	
	2	1	2	1	1	2	1	2	2	1	
	1	2	2	2	2	1	1	2	1	1	
Dyrene	2	2	1	1	2	2	2	3	1	2	3
	1	1	2	2	1	1	1	1	2	2	
	1	2	1	2	2	2	2	1	2	2	
	1	2	1	2	2	2	2	2	2	2	
	1	1	1	1	2	1	2	2	1	3	
	1	1	1	1	1	2	2	1	1	2	
	1	1	2	1	2	2	4	1	1	1	
	1	2	1	1	1	2	2	2	2	2	
	1	1	2	1	2	2	1	1	2	1	
	1	1	1	2	1	2	1	2	2	1	

[a]Disease ratings: 1 - no disease, 2 - 1 to 25% foliage infected, 3 - 26-50% foliage infected, 4 - 51 to 100% foliage infected.

[b]Trees rated 3 and 4 are not acceptable and worthless as Christmas trees.

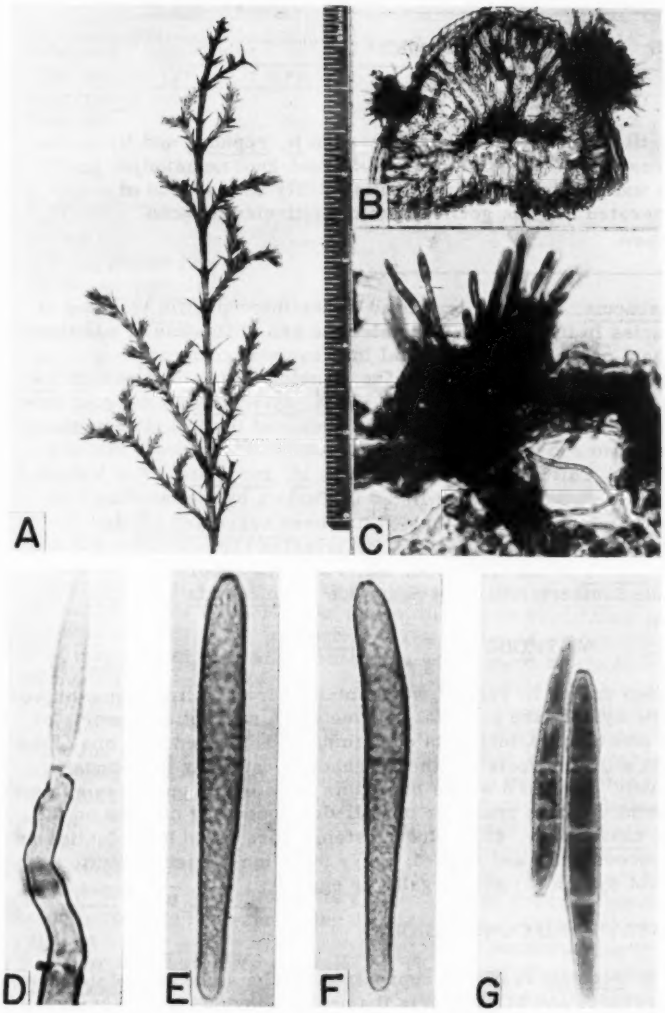

FIGURE 1. Cercospora thujina on Cupressus arizonica. A -- Blighted twig with stem cankers and needle infections. B -- Transverse section of needle with fascicles of conidiophores, approx. X289. C -- Fascicle of conidiophores with developing conidia and mycelium in leaf, approx. X397.5 D -- A conidium developing at tip of conidiophore, approx. X1350. E and F -- Typical conidia with minutely verrucose surface, approx. X1350. G -- Conidia stained with cotton blue to show septations, approx. X1200.

trees for each treatment. Three fungicides, Tribasic Copper Sulfate, maneb and 2,4-dichloro-6-(o-chloroanilino)-s-triazine (Dyrene) and an untreated control were used in the experiment. All of the fungicides were used at the rate of 2 1/2 pounds/100 gallons of water plus a wetter-sticker. Six applications were made at 14-day intervals, beginning June 3. The sprayed trees were given a disease rating of 1, 2, 3 or 4. Trees receiving a rating of 1 were free of disease; those with a 2 rating had from 1 to 25% of the foliage affected; those with 26 to 50% of the foliage affected had a rating of 3 and a rating of 4 indicated over 50% of the foliage infected.

Results: The results of the experiment are shown in Table 1. All of the fungicides used were highly significantly better than the untreated control. Although complete control was not obtained by fungicidal spray, Table 2 shows that fungicides greatly reduced the number of culls. The untreated control had 52% culls while Tribasic Copper Sulfate, maneb and Dyrene had 6, 2, and 3% respectively.

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RESPONSE OF NICOTIANA REPANDA, N. SYLVESTRIS, AND THEIR AMPHIDIPLOID HYBRID TO ROOT-KNOT NEMATODES

L. G. Burk and V. H. Dropkin[1]

Abstract

Nicotiana repanda and a fertile interspecific hybrid between N. repanda and N. sylvestris were grown in the presence of pure cultures of six root-knot nematodes. The response of the host plants was related to their potential utility as a source of resistance which may be incorporated into the germ plasm of cultivated tobacco through interspecific hybridization.

Cultivated tobacco, Nicotiana tabacum, has been improved by the interspecific transfer of disease resistance from certain species in the genus. The detection and utilization of additional sources of resistance is a regular part of the tobacco varietal improvement program.

This study was undertaken to measure the resistance of the amphidiploid hybrid, 4N (N. repanda x N. sylvestris)[2], and the parental species N. repanda and N. sylvestris to six root-knot nematodes. A comprehensive list of pathogenic plant nematodes prepared in 1954 (1) designates the six nematodes as four species and two sub-species. A recent report (6) suggests that the sub-species Meloidogyne incognita acrita Chitwood be considered as M. incognita. For the sake of simplicity, the nematodes used in this study have been listed in Table 1 by the familiar last name of their respective binomial or trinomial designation. It has been suggested (2) that N. repanda may be resistant to the Javanese root-knot nematode M. javanica (Treub) Chitwood and the peanut root-knot nematode M. arenaria (Neal) Chitwood, while another report (3) indicates that N. repanda may be immune to the Southern root-knot nematode M. incognita incognita Chitwood and some of its related strains.

METHODS

Inocula of the root-knot nematodes named in Table 1 were obtained from cultures maintained by the junior author. Seedlings of the hybrid, the parental species and a susceptible variety of tobacco, N. tabacum cv. 402, were planted in 4-inch pots containing soil infested with one of the six pathogens. Three series of tests were conducted in the greenhouse, starting in February, June, and November 1960, respectively. In 5 to 8 weeks the plants were examined for symptoms of root knot. The exact time was decided by the presence of well-developed egg masses on the roots of the susceptible "check," N. tabacum cv. 402. Root systems were rated for infection as follows: Massive infection with numerous galls and eggs-4; heavy infection-3; light infection-2; trace, one or two egg masses per root system-1; and no galls or eggs-0.

RESULTS AND CONCLUSIONS

Our data (Table 1) suggest that N. repanda responds approximately the same to Meloidogyne incognita acrita, M. javanica, M. arenaria and M. arenaria thamesi. Clayton, et al. (3) refers to the high resistance and immunity of N. repanda to M. incognita incognita. In an unpublished study, H. E. Heggestad and J. J. Grosso of Tobacco Investigations observed that plants of N. repanda and the N. repanda x N. sylvestris hybrid developed galls in the presence of M. incognita incognita after approximately 3 months of exposure.

Under our test conditions the polyploid hybrid grew with greater vigor than N. repanda. Therefore, we are unwilling to distinguish between the disease reaction of these host plants. The point of greatest significance is not that N. repanda had a lower index of infection than the check but that it supported multiplication of the four nematodes against which it was tested. Future breeding studies should reveal the utility of the low level of infection expressed by N. repanda. The behavior of N. repanda in combination with N. sylvestris offers some hope of success in this regard.

Tobacco breeding lines with high levels of resistance to M. incognita incognita and M. incognita acrita have been developed and tested (3, 4). Commercial varieties PD 611 and NC 95 have been jointly released by the South and North Carolina Experiment Stations, respectively, and the United States Department of Agriculture. This resistance was obtained intraspecifically by Clayton from a native South American tobacco, T. I. 706 (P. I. 116080). Therefore, the resistance which N. repanda may have against these root-knot species is of only passing interest.

[1]Geneticist and Nematologist, respectively, Crops Research Division, Agricultural Research Service, United States Department of Agriculture.

[2]Amphidiploid hybrid, courtesy of Dr. H. Hitier, Bergerac, France.

Table 1. Response of Nicotiana repanda, N. sylvestris and their polyploid hybrid to Meloidogyne species.

Host and date scored	Root-knot species and disease index					
	incognita	acrita	hapla	javanica	arenaria	thamesi
N. repanda						
7-27-60	---	1	---	1	---	2
1-19-61	---	1	---	0	1	0
Total plants (21)						
N. sylvestris						
7-27-60	---	3	---	4	---	3
1-19-61	---	2	---	2	2	2
Total plants (22)						
4N (N. sylvestris x N. repanda)						
4-4-60	1	1	0	0	---	---
7-27-60	---	1	---	0	---	0
1-19-61	---	1	---	0	0	0
Total plants (48)						
N. tabacum cv. 402						
4-4-60	3	3	3	3	---	---
7-27-60	---	4	---	4	---	3
1-19-61	---	2	---	2	3	3
Total plants (34)						

The response of N. repanda to M. javanica, M. arenaria and M. arenaria thamesi may be regarded either as a low level of susceptibility or resistance as compared with the response of N. tabacum cv. 402 or N. sylvestris. The choice of a proper designation for the disease response of N. repanda to root-knot nematodes will depend on its successful transfer to and utilization in cultivated tobacco varieties. The superior performance of the polyploid hybrid 4N (N. repanda x N. sylvestris) offers encouragement for further breeding studies. Aside from its contribution of vigor to the hybrid, the genetic role of N. sylvestris must be discounted in view of our results and those of Graham (5). It is known that M. incognita incognita and M. incognita acrita pose the greatest threat to tobacco production in the United States. However, M. javanica has occasionally been found as the predominant root-knot species in some tobacco fields of the southern States, especially Georgia and Florida. For this reason the resistance of N. repanda to M. javanica may be considered to have potential value, and efforts to transfer this germplasm to cultivated tobacco varieties are being made. The direct hybridization of 4N (N. repanda x N. sylvestris) x N. tabacum or its reciprocal has not been successful. An alternative method of bridge crossing has been under consideration. This method involves the hybridization of 4N (N. repanda x N. sylvestris) x N. sylvestris and backcrossing the nematode-resistant products of this hybridization to N. sylvestris. The end product of the first interspecific breeding pathway would yield an N. sylvestris with the nematode resistance of M. javanica. The second pathway would involve hybridizations of the modified N. sylvestris with N. tabacum. We hasten to add that this procedure is based on theoretical considerations only.

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THE EFFECT OF CONIDIAL CONCENTRATION ON PERITHECIAL FORMATION BY THE OAK WILT FUNGUS[1]

C. L. Fergus, W. J. Stambaugh, F. W. Cobb, Jr., and R. A. Schmidt[2]

Summary

A conidiation (spermatization) technique was studied to better evaluate its dependability and sensitivity. The effect of conidial concentration on the numbers of perithecia developed on a standardized colony was determined. A concentration of 22 conidia per 176 sq. mm of Ceratocystis fagacearum induced as many as three fertile perithecia. Thus, the method is relatively sensitive. However, the numbers of perithecia formed at the various conidial concentrations varied considerably between replicates. In addition, the suddenness with which an individual isolate may lose its ability to function as the (female) receptive thallus must be considered in using the method to determine the presence of the fungus in or on natural substrata.

INTRODUCTION

A special conidiation technique has been developed by Jewell (5) and Stambaugh, et al. (9). This technique has previously been referred to as a spermatization (4) process, but conidiation is a more accurate term (7) and will be used herein. It has been used to detect the presence of viable spores of Ceratocystis fagacearum (Bretz) Hunt in materials from which isolation by the usual culture methods ordinarily yield only fast growing bacteria and fungi that can outgrow the oak wilt fungus. The technique is based upon the fact that C. fagacearum is an hermaphrodite, with each isolate producing conidia that are able to act as male sex cells (as well as reproductive, germinating spores) and also the female ascogonium. However, each isolate is self-sterile but cross-fertile with isolates of the appropriate compatibility (4). Two compatibility types are known, and they have been arbitrarily designated as A and B. The material to be tested is suspended or ground up in sterile distilled water and then brushed onto A and B cultures. If perithecia mature and asci and ascospores develop, viable spores of C. fagacearum must have been present in the tested material.

The sensitivity of the technique has not been determined, although Stambaugh, et al. (9) showed that ground-up insects do not inhibit the production of asci and ascospores. These workers also reported that only two fertile perithecia developed per conidiated colony when a brush was dipped into a suspension of 4000 conidia per ml and then rubbed over the colony.

The conidiation technique has been used only to determine the presence of C. fagacearum in and on insects and insect feces (5, 6, 9). However, the method should be adaptable for use in determining the presence of the fungus in wounds, duff, soil, or other material.

Variability between isolates in their ability to act as a female (ascogonial) receptive thallus has been reported by various workers (1, 3, 8). Obviously this would affect the confidence one could place upon a negative result.

The following study was carried out to obtain a better evaluation of the dependability and sensitivity of the technique.

MATERIALS AND METHODS

Ten isolates of each compatibility were tested to obtain two cultures able to react well as receptive (female) thalli when conidiated with conidia of the appropriate compatibility. Culture number 2, isolated from a wilting chestnut oak (Quercus montana) in 1960, and culture number 11, isolated from a wilting red oak (Quercus rubra) in 1957, were found to be the best based on the type of mycelial development and subsequent perithecial production on Barnett's medium (1) in preliminary trials.

Petri dishes (9 cm diameter) containing 20 ml of Barnett's medium (1) were inoculated at four equidistant points with mycelium from 10 day-old colonies of each isolate. The dishes

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Table 1. The numbers of perithecia of C. fagacearum produced per colony after conidiation with varying numbers of conidia.

Numbers of conidia placed on one colony															
30,000		750		375		188		94		47		22		0	
F[a]	S[b]	F	S	F	S	F	S	F	S	F	S	F	S	F	S
2	0	5	2	8	1	16	2	6	0	1	0	2	0	0	0
4	2	30	1	12	2	8	0	3	0	1	1	2	2	0	0
5	0	2	1	35	4	6	0	1	0	0	2	1	0	0	0
17	1	23	6	5	0	24	1	8	0	0	0	2	0	0	0
27	1	25	1	2	1	15	2	6	1	0	0	0	0	0	0
49	1	18	2	0	0	9	1	3	2	0	0	3	0	0	0
7	2	21	0	5	2	0	0	6	1	8	1	2	0	0	0
28	2	16	1	1	0	4	0	4	0	4	1	0	2	0	0
14	0	5	3	2	0	6	1	16	1	0	1	1	1	0	0
10	1	4	.2	8	3	6	1	0	0	2	1	1	0	0	1
Totals 163	10	149	19	78	13	94	8	53	5	16	7	14	5	0	1

[a]F signifies fertile perithecia per colony.
[b]S signifies sterile perithecia (no extruded ascospores) per colony.

were incubated at 20° C for 12 days and then conidiated with varying numbers of conidia of the appropriate compatibility to induce development of fertile perithecia. The conidiating cultures were 7 day-old test tube cultures grown on Nutramigen agar. Nutramigen is a baby food manufactured by Mead Johnson and Company. The oak wilt fungus sporulates more abundantly on this medium than on any other tried (2).

Seven conidial concentrations were used (Table 1). A Fuchs-Rosenthal counting chamber was used to obtain a concentration of 200,000 conidia per ml. Portions of this original suspension were diluted to yield the more dilute conidial suspensions.

Glass van Tieghem cells, 15 mm in diameter, were sterilized in the autoclave and then aseptically placed on each colony to be conidiated. The cells were pushed down into the agar to the underlying glass plate to prevent the spore suspension from flowing out under the edges of the cells. Three drops of the conidial suspension were pipetted onto the 176 sq. mm of fungus colony circumscribed by a cell. The Petri dish was gently rotated by hand to disperse the conidial suspension over the circumscribed colony and then incubated 6 days at 20° C. Only colonies showing the best mycelial growth for perithecial induction were conidiated. Experience has shown that cultures with dense compact mycelium, producing many drops of amber fluid, will produce more abundant perithecia. There were ten replicates for each conidial concentration.

The conidiated cultures were examined by observing under a stereoscopic microscope at 36 diameters magnification. The numbers of perithecia extruding a white sticky ascospore mass were determined and recorded as fertile. Perithecia with necks that failed to open and which failed to extrude ascospores were also counted and recorded as sterile.

The two lowest conidial dilutions were checked for concentration and conidial germinability by pipetting one drop of each onto malt extract agar plates and determining total numbers of conidia and percentage germination after 18 hours at 20° C. Examination of the entire drop area under the compound microscope at 430 magnifications showed that one drop of the 312 conidia per ml concentration contained 15 conidia and one drop of 156 conidia per ml concentration contained 6 conidia. Assuming that one ml contains 20 drops, then 20 x 15 equals 300 conidia per ml, a remarkably close agreement to the 312 conidia actually counted.

RESULTS

Culture number 2 yielded perithecia at all conidial concentrations (Table 1). However, culture number 11, which had been selected just 3 weeks prior to the experiment as being able to act as a strong female receptor, yielded perithecia in any numbers only at the 750 and 375 spore concentrations. The result were so erratic that they are not presented here, but the maximum number of perithecia formed on any one area was 12; many produced fewer than 7.

In general, the more concentrated the conidial suspension, the greater the number of fertile perithecia developed. However, no direct relationship of concentration to numbers of perithecia was found. Considerable variation in the numbers of fertile perithecia within replicates of each conidial concentration was observed. The maximum number of fertile perithecia

was 49 in 176 sq. mm. The number of sterile perithecia, that is, perithecia that did not extrude ascospores by 6 days following conidiation, varied considerably on a sq. mm area basis.

DISCUSSION

The conidiation test is a useful technique for determining the presence of C. fagacearum on or in insects. Based on the experiments reported herein it is not quantitative. No direct relationship between concentration of conidia and the number of fertile perithecia induced was found. The numbers of ascogonia (female elements) produced in the culture determine the number of fertile and sterile perithecia to be produced by a colony. Therefore, the test as presently carried out cannot be quantitative, since a constant equal number of ascogonia cannot be produced. A method of culturing which will assure a constant and equal number of ascogonia in the culture to be used as the receptive thallus is to be desired. Even if this were possible, one would have to account for the numbers of conidia by counting matured perithecia on both A and B female receptive cultures since the conidia in one test source material would not necessarily be of only one compatibility. Wilson (10) has assumed that one conidium fuses with one ascogonium to induce one perithecium; however, since he failed to observe such fusion this assumption is based on analogy to other closely related Ascomycetes.

The conidiation test as carried out presently is quite sensitive. At the lowest concentration of 22 conidia in three drops, three fertile perithecia were induced. The mathematical possibility of each conidium coming in contact with a trichogyne (10), or the germ tube of the conidium contacting a trichogyne, within the 176 sq. mm area must be quite small.

The technique was slightly modified in this study to obviate possible effects from injury and also to be certain that all conidia applied to the colony remained on the colony. As normally performed, a brushfull of conidial suspension is brushed over the receptive thallus. This may result in breakage of hyphae and also some conidia may remain on the brush. The latter possibility would perhaps explain the low sensitivity of the test as previously reported (9).

Another problem is presented by these results. That of the sudden inexplicable loss of the ability of an isolate to function as a female receptive thallus. A worker using this technique should use at least four isolates as his female receptive cultures, preferably two of each compatibility type. In addition, a check colony on each plate at each test should be conidiated with appropriate conidia to ascertain that the colonies have not suddenly changed in their receptiveness.

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YIELD RESPONSE OF BRUSSELS SPROUTS TO SOIL FUMIGATION FOR CONTROL OF SUGAR-BEET NEMATODE, HETERODERA SCHACHTII

Bert Lear[1], R. H. Sciaroni[2], C. B. Atlee, Jr.[2], and W. H. Hart[3]

Summary

Three soil fumigation experiments were conducted over a 2-year period to control sugar-beet nematode, Heterodera schachtii, on Brussels sprouts. In two plots conducted on soils having pH 4, yield increases as high as 2.64 tons per acre were obtained with broadcast applications of Telone and D-D mixture. Row applications resulted in yield increases not significantly lower than the broadcast applications. Yields were increased as high as 2.37 tons per acre in one test conducted on soil of pH 6.

Poor growth of Brussels sprouts resulting in low yields often is seen in the cooler coastal counties of California where this crop is grown extensively. The soil acidity is about pH 4 in some of the soils of the area and some response has been obtained by the addition of lime to reduce manganese toxicity (1). Large numbers of the sugar-beet nematode, Heterodera schachtii, also are commonly found infecting roots of Brussels sprouts in this region. Many of the fields are uniformly infested probably largely due to the use of transplants grown in infested seed beds. It has been generally concluded that these infestations did little damage to this crop, or that if they were detrimental, little could be accomplished by way of control with soil treatment. The following experiments were conducted to determine the possible value of soil fumigation as a control of this nematode.

In 1959 a preliminary experiment was conducted in San Mateo County in which 20 and 40 gallons per acre of 95% 1,3-dichloropropene (Telone) alone and in combination with applications of dolomite lime were applied. The soil was pH 4.1 on these plots. Three replicate plots, each 10 feet by 100 feet (three rows) were included for each treatment. Brussels sprouts grown in seed beds fumigated with methyl bromide were transplanted 3 weeks after treatment. Three months after treatment soil samples were removed from each plot. Sugar beets were grown in these soil samples for 6 weeks and numbers of white females determined. Yields of Brussels sprouts were obtained by harvesting six times at 15-day intervals, following the normal practice of the grower.

In 1960 two additional experiments were conducted, one in San Mateo County on soil of pH 4 and the other in Santa Cruz County where the soil was about pH 6.0.

On the Santa Cruz plot, wilts caused by Verticillium spp. and Fusarium spp. had been determined as contributing to poor growth during the previous season. For this reason, chloropicrin (trichloronitromethane) was included alone and in combination with Telone. A wider range of doses, including row applications, were included in the San Mateo plot for Telone, 1,2-dichloropropane-1,3-dichloropropene (D-D mixture), and 1,2-dibromo-3-chloropropane (Nemagon). As in the preliminary experiment, soil samples were removed to determine degree of nematode control and yields were obtained by harvesting six times on the San Mateo plot and seven times on the Santa Cruz plot.

RESULTS

Yields for the preliminary experiment (Table 1) show statistically significant increases from all plots receiving an application of Telone. This was true for the first picking when increases of 0.93 tons to 1.49 tons were obtained. Plants growing on treated areas were ready for harvest 2 weeks earlier than on non-treated areas. There were no significant yield differences between the two doses used, although the combination of 40 gallons plus 4 tons of dolomite produced the highest yields. Nematode counts (Table 1) show the 40-gallon rate to be considerably more effective than the 20-gallon rate. As expected, the poorest nematode control was in the surface 6 inches.

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Table 1. Yields of Brussels sprouts and survival of nematodes from soil fumigation experiment for control of Heterodera schachtii. San Mateo County, 1959.

Treatment		First picking		Six pickings		Average no. females recovered on indicator plants grown in soil samples removed at following depths (in inches)		
Telone (gallons/acre)	Dolomite (tons/acre)	yield (tons/acre)	increase (tons/acre)	yield (tons/acre)	increase (tons/acre)	0 - 6	6 - 12	12 - 24
0	0	0.30	-	5.51	0	96	141	126
20	0	1.55	1.25	7.44	1.93	44	68	3
20	4	1.72	1.42	8.45	2.94	235	40	23
20	8	1.23	.93	7.76	2.25	96	143	40
40	0	1.23	.93	7.23	1.72	61	6	23
40	4	1.79	1.49	9.15	3.64	93	0	1
40	8	1.33	1.03	7.13	1.62	72	1	1
LSD 5%		0.54		1.40				
LSD 1%		1.00		1.93				

Table 2. Yields of Brussels sprouts and survival of nematodes from soil fumigation experiment for control of Heterodera schachtii. San Mateo County, 1960.

Treatment	Dosage (gallons/acre)	First picking		Six pickings		Average no. females recovered on indicator plants grown in soil samples removed at the following depths (in inches)			
		yield (tons/acre)	increase (tons/acre)	yield (tons/acre)	increase (tons/acre)	0 - 6	6 - 12	12 - 18	18 - 24
D-D	50	1.14	.70	8.25	2.64	4	1	8	176
D-D	30	1.27	.83	7.60	1.99	50	10	1	11
Telone	30	1.02	.58	7.43	1.82	6	0	1	17
D-D	40	1.15	.71	7.28	1.67	2	1	14	38
D-D (row)	13	.81	.37	7.12	1.51	81	86	49	114
Telone	40	1.01	.57	7.08	1.47	5	5	55	5
Telone (row)	13	.93	.49	7.07	1.46	39	4	13	213
Nemagon (row)	0.75	.59	.15	6.38	.77	111	21	229	137
Nemagon	1.5	.61	.17	5.79	.18	17	30	217	10
Check	0	.44	-	5.61	-	12	242	316	509
LSD 5%		0.32		1.00					
LSD 1%		0.44		1.38					

Table 3. Yields of Brussels sprouts obtained from one and seven pickings from soil fumigation experiment to control Heterodera schachtii. Santa Cruz County, 1960.

Treatment	Dosage (gallons/acre)	First picking yield (tons/acre)	First picking increase (tons/acre)	Seven pickings yield (tons/acre)	Seven pickings increase (tons/acre)
Telone	40	1.05	.40	10.00	2.37
Telone	30	1.31	.66	10.00	2.37
Telone + chloropicrin (row)	40 90 lb.	1.01	.36	9.49	1.86
Telone (row)	13	1.34	.69	9.24	1.61
Telone (row) + chloropicrin (row)	10 100 lb.	.96	.31	8.66	1.03
Chloropicrin (row)	90 lb.	.78	.13	8.54	.91
Check	0	.65	-	7.63	-
LSD 5%				1.37	
LSD 1%				1.88	

Yields for the 1960 experiment in San Mateo County (Table 2) show similar responses to treatment with Telone and D-D as in 1959. Increases ranged from 1.46 tons to 2.64 tons per acre. Row treatments resulted in yields not significantly different from the broadcast applications. The yield responses to treatment by Nemagon, however, were significantly less than with either D-D mixture or Telone. Even though the broadcast application of 1.5 gallons resulted in better nematode control than the row application of 0.75 gallons, yields were less than for the row application. This suggests that Brussels sprouts are sensitive to Nemagon at the doses employed. Nematode counts (Table 2) show that all broadcast applications of D-D and Telone were very effective in the top 18 inches. Poorer control was obtained at the 18 to 24 inch depth.

From the Santa Cruz plot, yield increases were the same for the 30- and 40-gallon rates for Telone (Table 3). Chloropicrin in combination with Telone did not improve yields over Telone alone. Chloropicrin alone at 90 pounds per acre in the row resulted in the lowest yields of the treatments used.

DISCUSSION

Brussels sprouts are expensive to grow. Based on a yield of 4 1/2 tons per acre, Parsons and Sciaroni (2) list total production costs at 7.7 cents per pound. During the past 2 years, growers of Brussels sprouts have operated under a marketing order that establishes a fixed tonnage that may be processed from each grower. Increases in yields resulting from sugarbeet nematode control make it possible for the growers to fill marketing quotas by farming considerably fewer acres. This will permit growers to include other crops in rotation with Brussels sprouts. Rotations are of great benefit in the control of certain diseases, including club root, of this crop.

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GNOTOBIOTIC TECHNIQUES AND THE STUDY OF RADOPHOLUS SIMILIS ON CITRUS[1]

E. P. DuCharme and R. W. Hanks

Ordinary techniques for studying the effects of a nematode parasite on the roots of a plant are not suitable for determining the specific effects of Radopholus similis (Cobb 1893) Thorne 1949 on rootlets of citrus or for distinguishing such effects from those induced by other organisms, because of the presence of contaminating organisms. Consequently, a procedure was devised to grow citrus seedlings in a gnotobiotic environment, that is, an environment free of all other organisms except for those selected. The procedure and its uses are described in this paper.

The terminology used was proposed by Dougherty (3) for describing cultures containing known species. Dougherty defined gnotobiology as "the science of growing organisms of one species under conditions where their environment contains either no, or only known other species."

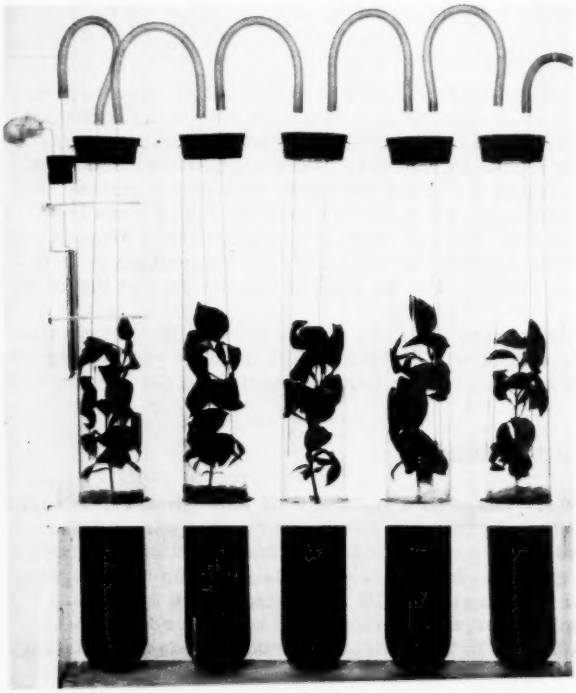

FIGURE 1. Seedlings of grapefruit (Citrus paradisi) in gnotobiotic culture tubes.

A review of literature reveals the results of experiments that have been made with axenic seedlings of apple (12), citrus (5), cowpea (1) and peach (10) cultured on synthetic media in association with plant parasitic nematodes. Generally the cultures were maintained for relatively short periods. One apparatus, however, was designed especially to maintain axenic cultures of seedlings for several months and to culture plant tissues for indefinite periods (13). All the methods described had the limitation that the systems could be invaded by contaminating microorganisms and were not designed to maintain plants for more than 1 year.

Effective procedures that have been proposed for axenizing free living and plant parasitic nematodes range from repeated washings in sterile water (16) to treatments with chemical disinfectants. Chemicals found to be effective and used were hydrogen peroxide (7, 15), mercuric chloride (1, 2, 6, 11), merthiolate (4), sodium hypochlorite (2, 7, 9), 8-hydroxyquinolin sulfate (2, 4, 5), ethoxy ethyl mercury chloride (2, 8, 10, 14), and antibiotics (4, 8, 14).

The method developed for establishing monoxenic cultures of Radopholus similis on Citrus paradisi (grapefruit) consists of three parts: a) preparation of the physical environment; b) preparation of axenic host plants (grapefruit seedlings); c) preparation of axenic parasites (R. similis).

The axenic physical environment was contained in large pyrex test tubes, 65mm x 500mm (Fig. 1), filled to a depth of 175mm with Lakeland fine sand subsoil moistened with sufficient nutrient solution to approximate field capacity. The culture tubes were closed with rubber stoppers fitted with two glass tubes extending nearly to the surface of the soil. The exposed ends of all tubes were capped with cotton and the closed culture tubes were steam sterilized for 30 minutes at 15 pounds' pressure.

Fresh grapefruit seeds were soaked in 0.25% sodium hydroxide for 20 minutes to remove pectic substances from the outer seed coat. The tough outer seed coat was next removed from the seed. The seeds, still protected by the inner integument, were placed in 1% aqueous so-

[1]Florida Agricultural Experiment Stations Journal Series, No. 1296.

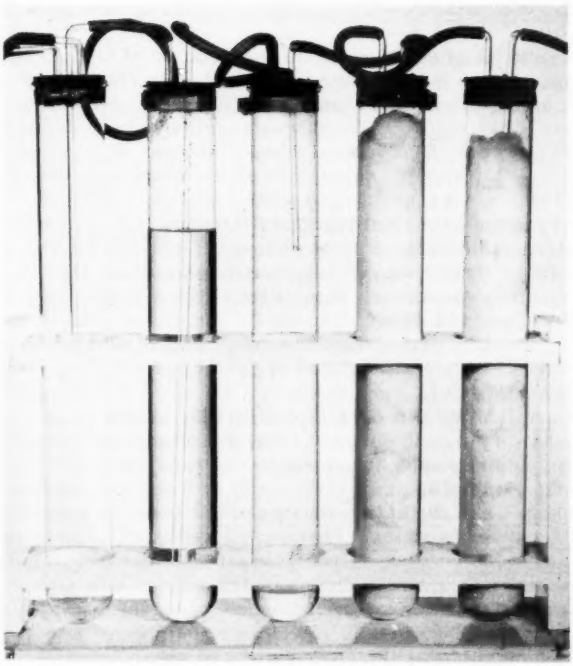

FIGURE 2. Equipment for filtering, moistening, and distributing air to gnotobiotic culture tubes.

lution of 8-hydroxyquinolin sulfate or in mercuric chloride, 1000 ppm. The inner integument was then carefully separated from the seed and the seed rinsed in 0.5% sodium hypochlorite. The treated seeds were sown on sterile 1.5% water agar in Petri dishes. Seeds treated in this manner germinated in 3 to 5 days and seedlings 1 to 2 weeks old were large enough for use in gnotobiotic cultures.

Vigorous female nematodes were selected from parasitized citrus rootlets and placed in 1% aqueous solution of 8-hydroxyquinolin sulfate for 10 minutes and then washed in 0.2% aqueous streptomycin sulfate. Burrowing nematodes could not be transferred directly to citrus seedlings from 8-hydroxyquinolin sulfate because this chemical is toxic to the roots. Streptomycin sulfate is a suitable rinse since it is not toxic either to burrowing nematodes or to citrus roots. The effectiveness of this method for freeing burrowing nematodes of microorganisms was verified by plating some of the treated nematodes on nutrient agar.

The monoxenic cultures of citrus and burrowing nematodes were established after the various components had been prepared. Seedlings with straight roots 1/2 to 1 inch long were transferred to sterile 1.5% water agar in other Petri dishes or were planted in the large culture tubes. The seedlings were inoculated either by placing one or more of the treated burrowing nematodes next to the root tips or by pipetting sterile rinse containing axenic nematodes onto the root tips. For the large culture tubes it was better to plant seedlings already infected with R. similis than to inoculate the seedlings after they were planted in the culture tubes.

After assembly, five to ten of the gnotobiotic culture tubes were connected in series by attaching sterilized rubber tubing to the glass tubes protruding through the rubber stoppers of the culture tubes (Fig. 1). Ventilation of a series of culture tubes was maintained by a continuous flow of compressed air successively passed through tubes of sterilized cotton to remove foreign matter and through sterilized water to moisten the filtered air (Fig. 2). The sterilized air, distributed from a manifold and passed through the gnotobiotic culture tubes, was bubbled through small test tubes of sterile water at the end of each train of culture tubes to observe the rate of air flow through the system. Screw clamps on the sterilized rubber tubing leading from the manifold controlled the rate of air flow through each system of gnotobiotic culture tubes. Four series of culture tubes could be supplied with air from one set of filters and manifold. All items used in assembling a series of gnotobiotic culture tubes were sterilized.

With this method it was possible to study penetration of burrowing nematodes and subsequent development of lesions in citrus rootlets and to observe the effects of R. similis on the whole plant under gnotobiotic conditions (5). Axenic citrus seedlings have been grown for 14 months in these culture tubes.

Gnotobiotic techniques are being used now to study population dynamics of R. similis, the influence of temperature on the reproduction and life cycle of R. similis, the effects of soil fungi on the roots of citrus seedlings and the effects of R. similis alone and in combination with other selected microorganisms on citrus seedlings. The length of time that gnotobiotic cultures on citrus and other organisms can be maintained seems to be limited only by the size of the culture chamber. A plastic film culture chamber 28 inches wide x 48 inches long x 28 inches high is being used to house axenic grapefruit seedlings planted in soil contained in crocks of 1-gallon

capacity. This culture chamber is ventilated constantly with moistened sterile air and provided with sufficient light to maintain plant growth.

Gnotobiotic techniques of this type permit the study of the biotic association of a plant and a parasite in a physical environment comparable to that in nature without the interference of other organisms. These methods may be useful to study the interaction of a host and pathogen in other plant diseases.

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NEW HOSTS FOR CERCOSPORA THUJINA PLAKIDAS

Charles S. Hodges[1]

Cercospora thujina Plakidas was first described in 1945 as causing a blight of leaves and branchlets of oriental arborvitae (Thuja orientalis) and possibly Italian cypress (Cupressus sempervirens) in Louisiana and surrounding States (1). Wilson (2) in 1961 reported that this fungus caused a similar disease of Arizona cypress (C. arizonica) in Arkansas.

In 1960 a serious needle blight disease was noted on 1-year-old seedlings of eastern redcedar (Juniperus virginiana) in a forest nursery near Saint George, South Carolina. C. thujina was found sporulating profusely on dead needles of infected plants. The distinguishing symptom of the disease was a browning of the needles on the lowest branches adjacent to the stem. Infection gradually progressed upward and outward until only the branch tips remained green on severely affected plants. These symptoms are identical with those described by Wilson (2) for Arizona cypress. This disease is easily distinguished from the common Phomopsis blight, Phomopsis juniperovora Hahn, present on Arizona cypress and redcedar seedlings in more forest nurseries, which kills the tips of the branches. Arizona cypress in a windbreak adjacent to the nursery beds was also severely infected with C. thujina and possibly served as the source of inoculum for the seedlings.

The blight was also found on redcedar seedlings growing in a forest nursery near Macon, Georgia. Adjacent to this nursery was an experimental Christmas tree plantation including the following species: Cupressus arizonica, C. funebris, C. lusitanica, C. macrocarpa, C. sempervirens, Juniperus virginiana, Cedrus atlantica, C. deodora, and Pinus sylvestris. Typical blight symptoms and fruiting bodies of Cercospora thujina were observed on all species of Cupressus with the exception of C. funebris. The disease was particularly severe on C. sempervirens with all trees at least 50% defoliated. C. macrocarpa was also severely affected. One tree of J. virginiana was lightly affected. The other tree species present were not affected.

Several plants of Chamaecyparis pisifera var. squarrosa, growing as ornamentals in the vicinity of Raleigh, North Carolina, were found showing typical blight symptoms and with Cercospora thujina sporulating on dead needles.

Single-spore isolates of C. thujina from Chamaecyparis pisifera, Juniperus virginiana seedlings from South Carolina and Georgia, Cupressus arizonica from Georgia, and Thuja orientalis from Arkansas (obtained from Dr. Charles Wilson, University of Arkansas) were compared in pure culture on potato-dextrose medium and found to be essentially identical with respect to growth and colony appearance. The isolates from J. virginiana from South Carolina and T. orientalis from Arkansas were pathogenic to J. virginiana, C. arizonica, and T. orientalis in inoculation studies carried out using techniques similar to those used by Wilson (2). Inoculation studies were not conducted on the remaining species reported as hosts.

It is believed that Cupressus lusitanica, C. macrocarpa, Chamaecyparis pisifera, and Juniperus virginiana have not been reported as hosts for Cercospora thujina. Four genera of the Cupressaceae are now known to be hosts to this fungus: Chamaecyparis, Cupressus, Juniperus, and Thuja. The reported range of C. thujina now extends from Texas to North Carolina and this fungus undoubtedly occurs throughout the southern United States.

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FIRST REPORT OF ROOT-KNOT NEMATODES ON GRAPE-HYACINTH[1]

D. P. Taylor and R. L. Norgren[2]

The bulb and stem nematode, Ditylenchus dipsaci (Kuehn, 1857) Filipjev, 1936, observed on Muscari botryoides (1) and Muscari sp. (2) is the only nematode parasite reported on grape hyacinth.

FIGURE 1. Muscari armeniacum infected with Meloidogyne hapla. Bulblet on the right. Root-knot gall with egg masses at lower left.

Several flats of M. armeniacum grown from seed at the University of Minnesota grew poorly and were mildly chlorotic. Many root-knot nematode galls (Fig. 1) were found on the roots. Specimens dissected from galls were identified on the basis of perineal patterns as the northern root-knot nematode, Meloidogyne hapla Chitwood, 1949. This is the first report of parasitism of a species of Muscari by a root-knot nematode.

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A SIMPLE SUBSTITUTE FOR A BAERMANN FUNNEL

A. F. Schindler[1]

During the course of a test on the effects of soil fumigants on nematodes and on rose plants when applied as preplant treatments and on established outdoor rose plants (2) it was necessary to collect and wash up to 64 soil samples and extract the nematodes in 1 day. To simplify the process and to conserve space a simple substitute for the Baermann funnel was devised (1).

As shown in Figure 1, a circular hole 3 3/8 inches in diameter was cut in a 16 x 5 x 3/4 inch board. To make a plunger a 3/4 x 5 inch dowel was fastened to the 3 1/4 inch diameter piece removed from the board. The required 4 1/4 inch diameter circles were cut from aluminum window screening. Each circle of screening was placed over the hole in the board and forced through it by means of the plunger. This formed the screen into a dish shape with a 3 1/4 inch diameter bottom and sloping sides 1/2 inch high.

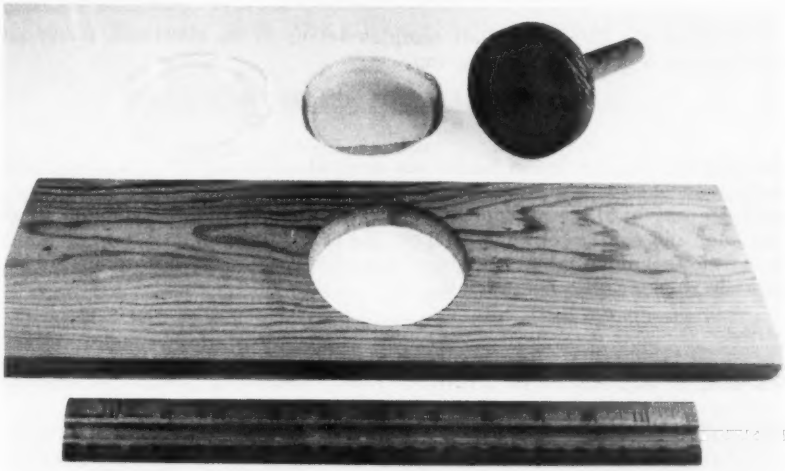

FIGURE 1. Equipment and material used in making the substitute Baermann apparatus.

The screens were placed in 3 3/4 inch Petri dish tops, the sloping sides serving to hold the screens above the bottom of the dishes. Two-ply Kleenex tissues[2], 8.5 x 9.7 inches, were cut into four equal parts, each part serving to cover the surface of one screen. Water was poured onto the tissue surface until it was moistened up to at least the edges of the aluminum screen. All excess tissue was gently torn from the upper edge of the screen, since otherwise water drained from the dish by capillary action.

The screens with the moist tissue were set in the Petri dishes and the nematode suspensions from the washing process added. The water and the sievings from the screen used in washing each soil sample was kept at or below 50 ml, the approximate capacity of the Petri dish. When the volume of the suspension was more than 50 ml the nematodes and debris were allowed to settle and some of the water was then decanted. The water and sievings were then poured directly onto the surface of the moist tissue and, if necessary, additional water was added until the level of the water was slightly above the tissue surface. After 24 hours the screens were removed from the dishes. The water and nematodes in the Petri dishes were poured into 50-ml beakers and allowed to settle. Next, 30 to 35 ml was decanted and the remaining nematode suspensions were poured into Syracuse dishes for examination and counting.

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[2]The mention of commercial products anywhere in this paper does not imply that they are endorsed or recommended by the Department of Agriculture over others of a similar nature not mentioned.

FIGURE 2. Thirty-two soil samples set-up in the substitute Baermann apparatus on a portion of a desk-top.

The substitute for the Baermann funnel has several advantages: 1) a large number of samples can be processed in a minimum amount of space (Fig. 2); 2) the materials are readily available and inexpensive; 3) the equipment necessary for processing a number of samples is compact, portable, and easily set up away from the laboratory.

In addition, the technique might also be of use in high school laboratories or other institutions interested in working with nematodes.

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OAK WILT IDENTIFIED IN TEXAS

O. J. Dooling[1]

The causal agent of oak wilt, Ceratocystis fagacearum (Bretz) Hunt, has been identified on two red oak (Quercus sp.) specimens from Dallas, Texas. The author has not observed these two trees; specimens were submitted by Robert R. Cunningham, an arborist of Dallas, on May 26, 1961.

The discovery of oak wilt in Dallas is approximately 200 miles to the south of previously reported occurrences. The disease has been reported from Crawford and Logan counties in Arkansas[2].

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DUTCH ELM DISEASE FOUND IN MINNESOTA[1]

D. B. Schroeder and D. W. French

In March 1961, the Dutch elm disease fungus, Ceratocystis ulmi (Buis.) C. Moreau, was isolated from an American elm, Ulmus americana, in St. Paul, Minnesota. This tree had been dead for approximately 6 months at the time isolations were made. As soon as the fungus was identified, the tree was felled, hauled away, and burned. Subsequently, Dr. J. C. Carter, of the Illinois Natural History Survey, confirmed the identification of the fungus. The native elm bark beetle, Hylurgopinus rufipes (Eichh.), was present under the bark of the tree. In July the smaller European elm bark beetle, Scolytus multistriatus (Marsh), was found in a city dump in St. Paul by Gerald Beach, staff member of the Minnesota State Division of Plant Industry.

Several elm trees five years old were inoculated, in the greenhouse, with the isolate of the Dutch elm disease fungus from St. Paul and with a known isolate of C. ulmi. Typical wilt symptoms appeared in all of the inoculated trees and the isolate from the dead tree in St. Paul caused symptoms similar to those caused by the known isolate. C. ulmi was isolated from all of the inoculated trees. All of these inoculated trees were destroyed by autoclaving as soon as wilt symptoms occurred.

No other cases of Dutch elm disease have been found in Minnesota, but samples from dead and dying elm trees are being collected and cultured.

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[1]Paper No. 4672, Scientific Journal Series, Minnesota Agricultural Experiment Station.

BOOK REVIEW

"ARE YOU YOUR GARDEN'S WORST PEST?" by Cynthia Westcott, Doubleday, New York City. 305 pages. 1961. Price $4.50.

Of Dr. Westcott's very practical books for the home gardener none can be more useful than this one. In "Are You Your Garden's Worst Pest?" she mixes technical knowledge and common sense in palatable "doses" of advice beneficial to the most expert gardener.

As anyone familiar with the work and writings of "The Plant Doctor" would expect, her new book contains the latest recommendations -- in all, nearly a thousand -- for chemical control of the diseases and insects attacking garden plants.

But this chemical control takes up only one of seven important chapters in her new book. Dr. Westcott also discusses the need for diversifying garden plantings so that a pest outbreak won't cause wholesale destruction. She calls attention to the danger of planting "alternate hosts," that is, different kinds of plants that some rusts need to complete their life cycle.

She talks about plant quarantine regulations in a way to make them understandable and, we hope, in a way to instill more patience in shippers who must sometimes take special efforts to comply with them.

Dr. Westcott tells how and when to prune trees and shrubs, especially roses. She discusses plant culture -- planting, mulching, fertilizing, and watering. She puts sanity into garden sanitation, advising gardeners to temper "clean up" with other good conservation practices.

Dr. Westcott precedes her chapter on spray recommendations with two that give it perspective -- "Is This Spray Necessary?" and "Spare Your Friends." The first cautions against a gardener's normal desire to counter every problem with a dust or spray. It specifically points out the fallacy of applying protective fungicides to diseased plants. The second reminds us that some garden insects are beneficial and should be protected. Dr. Westcott describes these and their habits.

As a bonus, Dr. Westcott includes an appendix of three useful lists. One of these is on garden pesticides, their common or trade names, their compatibility with one another, and the precautions to be taken in their use. The optimum pH (soil acidity) range for most garden plants constitutes the second list, and State and federal plant quarantine regulations the third. -- PAUL R. MILLER

CORRECTION

REPORTER, June issue (Volume 45, Number 6). On page 457, the second line in the Summary should read "and bananas only are infected under field conditions." The Reporter regrets the misplacing of the word only.